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OTORHINOGENIC MENINGITIS

A REPORT OF FIFTY-EIGHT CASES

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Meningitis is still the most frequent fatal complication of suppurative disease of the ears and sinuses. In reviewing 160 consecutive cases of meningitis in patients seen in consultation from Jan. 1, 1938 to April 30, 1941 at the Harlem Hospital, we found an otorhinogenic focus in 58, or 36.2 per cent. Because of this fact, we feel justified in reporting our experience with this series of cases. In presenting some of the literature on the subject, it is not the intention to mention every possible clinical detail for the sake of completeness; it is taken for granted that the reader is familiar with most or all of those points and facts that are now commonly accepted knowledge. We shall discuss only points in anatomic pathways, diagnosis and treatment, in order to bring into juxtaposition and appraisal the differing opinions for the purpose of presenting a comprehensive picture of the disease and of arriving at some logical procedure in treatment.

Rhinogenic intracranial complications result most frequently during the course of an acute lesion, in contradistinction to otogenic intracranial complications, which are said to occur most often during the course of chronic otitis media. In our series (table 5) if we arbitrarily classify as chronic all cases in which the duration of symptoms was more than one month and include those in which the history was doubtful, only 11 could be tabulated as chronic.

Our cases have been analyzed as to suspected focus, age, race and sex. Table 1 shows that in 38 of 58 cases the condition was otogenic and in 20 rhinogenic. The disease was predominantly one of adult life, as only 9 of the 38 cases of otogenic meningitis and 5 of the 20 cases

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of the rhinogenic type occurred before the age of 16 years. Table 1 discloses a total of 30 females and 28 males; otogenic infection was more prevalent in females and rhinogenic infection in males. The total number of Negro patients was 47 and that of white patients 11, but this factor cannot be properly evaluated, since a great proportion of the patients in the Harlem Hospital are Negroes.

The development may be insidious and the condition not suspected until it is already dangerously advanced. McCaskey [1] stated that not infrequently an infection in the ear which is throwing out organisms to produce meningitis is overlooked, or more often considered nonsurgical, because of lack of clinical or roentgenographic evidence.

TABLE 1.—*Analysis of Fifty-Eight Cases of Meningitis with Otogenic or Rhinogenic Focus* *

Age	Suspected Focus		Race				Sex			
			Otogenic		Rhinogenic		Otogenic		Rhinogenic	
	Otogenic	Rhinogenic	N	W	N	W	M	F	M	F
Less than 1 year	4	1	30	8	17	3	17	21	11	9
1- 5	3	3								
6-10	2	1								
11-15	0	0	Total negroes 47				Total males 28			
16-20	3	1	Total white persons 11				Total females 30			
21-25	1	0								
26-30	2	1								
31-35	3	5								
36-40	7	2								
41-45	3	2								
46-50	1	0								
51-55	3	2								
56-60	3	2								
61-65	1	0								
66-70	1	0								
71-75	1	0								
Total	38	20								

* Of 160 consecutive cases of meningitis occurring from Jan. 1, 1938 to April 30, 1941, 58, or 36.2 per cent, had an otorhinogenic focus.

Eagleton [2] stated that nearly 50 per cent of all cases of rhinogenic meningitis follow intranasal operations and that the meningitis is but a recrudescence of a former meningeal infection, as all the patients have a history of previous headache, and often attacks of vomiting. Such symptoms should be a warning to the surgeon to prepare the patient adequately by appropriate measures, such as vaccine therapy and administration of sulfanilamide or a derivative, and he advised the performance of a lumbar puncture before subjecting the patient to any nasal operation on diseased tissue proximate to the dura.

1. McCaskey, D. H.: Treatment of Otitic Meningitis Due to Streptococcus Infection by Surgery and Sulfanilamide, Surg., Gynec. & Obst. **68**:377-389 (Feb.) 1939.

2. Eagleton, W. P.: Intradural Conditions in Relation to Rhinology and Otology, Arch. Otolaryng. **32**:54-120 (July) 1940.

Brunner [3] reported a case of fatality from meningitis after removal of polypi from the right side, the infection traveling to the dura along the sheaths of the olfactory nerve.

Imperatori [4] advised against operation on the septum or removal of polypi in the presence of acute infection. He reported 17 cases of rhinogenic meningitis. In 7 cases it followed intranasal operation on the sinuses and in 8 complicated disease of the paranasal sinuses. Staphylococci were cultured in 9 cases, streptococci in 5 and pneumococci in 2.

Fehr [5] analyzed 417 cases of meningitis and found that in 20, or 4.8 per cent, the condition could be traced to previous fracture of an accessory sinus or the petrous pyramid. In 5 of the cases of otogenic meningitis a transverse fracture of the petrous pyramid was demonstrated. In 1 case meningitis followed acute otitis six months after the accident and in another case three years after. Meningitis of otogenic origin develops more easily in persons with a history of previous fracture of the petrous pyramid because such fractures heal by fibrous union, which leaves thus a permanent possible pathway for infection of the ear to reach the meninges.

In only 8 of our series could meningitis be attributed to former trauma and in 3 to former operative trauma.

Pneumococcic infection is by far the most common, as shown in table 4. It appeared in 27 of the 38 cases of otogenic type and in 10 of 20 cases of the rhinogenic type, thus representing almost 75 per cent of cases of the former and 50 per cent of cases of the latter. Of the cases in which the disease was attributable to infection of the ear, Pneumococcus type III was present in 9; next in order of frequency were types XX, IV, VI, VIII and XXIII. The streptococcus was isolated in 5 cases. In the cases of rhinogenic type pneumococcic types II and III were predominant, and streptococci were found in 2 cases. In evaluating the tabulation it must be stated that a very large number of these cases were referred to us from the pneumonia service of Dr. J. G. Bullowa. The others were from the pediatric service of Dr. M. Gleich.

Otorhinogenic meningitis represents a protective inflammatory reaction, which may result from infection in the middle ear, the mastoid, the petrous pyramid or the labyrinth or in the accessory sinuses of the nose, particularly the sphenoid and ethmoid.

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3. Brunner, H.: Zur Pathologie und Klinik der rhinogenen Meningitis, Ztschr. f. d. ges. Neurol. u. Psychiat. **157**:92-99, 1937.

4. Imperatori, C. J.: Differential Diagnosis of Suppurative Meningitis Caused by Paranasal Sinus Disease with Some Suggested Prophylactic Measures, Laryngoscope **47**:300-310 (May) 1937.

5. Fehr, A.: Zur Behandlung von Schädelbasisbrüchen bei Komplikationen von Seiten der Nasenbenhöhlen und des Ohres, Helvet. med. acta **4**:637-641 (Nov.) 1937.

TABLE 2.—*Data on Cases of Meningitis Following Operative Trauma*

Organism	Operation	Result
Pneumococcus VIII	Myringotomy	Death
Pneumococcus VIII	Bilateral transantral ethmoidectomy	Recovery
None	Bullet in mastoid removed; meningitis developed; operation, with removal of bony spicules	Recovery

TABLE 3.—*Data on Cases of Meningitis Due to Former Trauma*

Organism	Trauma	Result
Pneumococcus		
III	Fracture through maxilla and hard palate	Death
IV	Fracture through frontal sinus	Death
IV	Fracture of skull with maceration of brain	Death
IV	Fracture through frontal sinus	Death
XX	Fracture of skull into orbit and frontal sinus	Death
Streptococcus		
Beta	Fracture of maxilla, zygoma and hard palate	Death
Beta	Fracture of ethmoid bone and skull; possible fracture of petrous pyramid	Recovery
None	Bullet in mastoid	Recovery

TABLE 4.—*Organisms Involved in Cases of Otorhinogenic Meningitis*

	Otogenic Meningitis, No. of Cases	Rhinogenic Meningitis, No. of Cases
Pneumococcus type		
I	1	0
II	0	0
III	9	2
IV	2	2
V	2	0
VI	2	0
VII	1	0
VIII	2	1
IX	0	0
X	0	1
XI	0	0
XII	1	0
XIII	0	0
XIV	0	1
XV	0	0
XVI	0	0
XVII	1	0
XVIII	1	0
XIX	0	1
XX	3	0
XXI	0	0
XXII	0	0
XXIII	2	0
XXIV	0	1
XXV	0	1
Unknown	1	0
Streptococcus		
Beta	3	2
Gamma	2	0
Meningococcus	0	4
Haemophilus influenzae	0	3
Miscellaneous organisms	..	1
Bacillus proteus	1	..
Bacillus Friedländer A	1	..
Bacillus xerosis	1	..
Bacillus pyocyaneus	1	..
Bullet	1	..
Total	38	20

Inflammatory erosion of the cerebral bony wall at some point, such as the tegmen tympani or tegmen antri, in the region of Trautmann's triangle or the roof of the ethmoid or sphenoid sinus, is said to be the most frequent lesion. This results from the action of osteoclasts stimulated by activity of the toxins of the bacteria and by the absorption of calcium salts through the agency of the body fluids. Except in cases of traumatic origin we have not been able to substantiate this observation.

PATHWAYS OF INFECTION

Infection of the meninges may result when micro-organisms enter the arterial stream from the accessory sinuses or the temporal bone, are carried to all parts of the body and are arrested in the small pial arteries. When infection around the veins of the mucosa occurs, bacterial toxins transfuse through the vessel wall and, injuring the endothelial lining of the vessel, produce a thrombus, which becomes infected and advances by retrograde extension to cause meningitis.

There is no direct continuity between the lymphatics of the peripheral parts and the leptomeninges of the brain,[6] but infection in the perivenous lymph spaces is carried to the lymph spaces around the larger veins and from these spaces the bacteria enter the lumen of the vein to reach the leptomeninges.

An intermediate pathway of infection may exist, as in meningitis which follows brain abscess, or there may be present thrombophlebitis of the cavernous sinus, the latter being, according to Turner and Reynolds,[7] a rather frequent occurrence. Veins from the lining membrane of the sphenoid sinus enter the haversian canals in the osseous wall of the cavity and pass directly into the cavernous sinus. The anterior and posterior ethmoid veins enter the superior ophthalmic vein and so drain into the cavernous sinus. Infection in the carotid plexus, which receives branches from the lining membrane of the tympanic cavity, infection in the petrosal sinuses, the emissary vein of the mastoid and later the cavernous sinus are other intermediate pathways.

Brunner [8] stated that the meninges can become infected by invasion through one pathway or through several pathways simultaneously, in which event the meningitis is quickly generalized or is circumscribed for only a very short period. This is particularly true if the meningitis originates in the anterior or the middle fossa of the skull. This multiple

6. Goodyear, H. M.: Indications for Surgery in Meningitis Secondary to Disease of the Middle Ear and of Nasal Sinuses: Reservations in Early Surgical Intervention, Laryngoscope **49**:102-105 (Feb.) 1939.

7. Turner, A. L., and Reynolds, F. E.: Intracranial Pyogenic Diseases, London, Oliver & Boyd, 1931.

8. Brunner, H.: Pathology and Treatment of Otitic and Rhinogenic Meningitis, Illinois M. J. **77**:57-63 (Jan.) 1940.

invasion is most frequent in cases of otitic meningitis and in cases of rhinogenic meningitis in which the infection travels along the sheaths of the olfactory bundles.

Infection of the meninges may take place through the ductus endolymphaticus, which connects the endolymphatic space of the labyrinth with the dura in the posterior cranial fossa, through the aqueduct of the cochlea, which connects the perilymph fluid lying between the bony and the membranous labyrinth directly with the spinal fluid, and along the pia-arachnoid, which covers the auditory and vestibular nerves in the internal auditory meatus down into the labyrinth itself.

Kramer and Som [9] in a series of cases in which microscopic examination was made, were also able to demonstrate evidences of spread to the meninges as a result of osteomyelitis and osteitis, lymphatic extension via perineural sheaths, lymphatic extension via perivascular lymph channels, vascular spread through perforating vessels to the cavernous sinus, direct invasion of the meninges through congenital dehiscence and spread through a persistent craniopharyngeal pouch. They cited the work of Zwillinger, who injected dye into subarachnoid spaces and filled the perineural lymph spaces in the olfactory mucosa. The dye was further suffused into the superficial lymphatic network of the olfactory mucosa. They reported 3 cases in which progressive erosion of the compact bone of the roof of the ethmoid and sphenoid sinuses resulted from infection, so that there was a direct continuity between the sinus and the dura. In their cases of osteomyelitis they found an instance of acute exacerbation of a chronic submucosal abscess, with extension to the bone. In 1 case there were perineural infection in the smaller filaments of the olfactory nerve in the mucosa below the cribriform plate, dilatation of the perineural spaces, polymorphonuclear infiltration around the layer of nerve fibers and extension of the infiltration endocranially through the cribriform perforations to the subarachnoid spaces. They made the point that if the perineural infection were secondary to the infection in the subarachnoid space, the infiltration would be limited to the area above the cribriform plate and would not involve the small filaments in the olfactory mucosa.

In only a small number of cases can a microscopic infectious lesion in the paranasal sinuses be demonstrated as a probable focus of infection. Microscopic bony defects, when they occur and produce an avenue for infection, are invariably traumatic in origin and most often result from intranasal operation.

That part of the ethmoid capsule which is adjacent to the dura is made up of lamellae of compact bone. There is no bone marrow in these

9. Kramer, R., and Som, M. L.: Intracranial Pathways of Infection from Diseases of the Sphenoid and Ethmoid Sinuses, Arch. Otolaryng. **32**:744-770 (Oct.) 1940.

olfactory laminas. Compact bone does not harbor infection, and therefore meningitis from ethmoiditis of the anterior and middle group of cells is practically nonexistent.

Although dehiscences are frequent in the lamina papyracea and microscopic congenital dehiscences are not uncommon in the sphenoid bone, meningitis rarely occurs (except in cases of operative trauma) because the external layer of the dura is covered by mucous membrane, which is the protective mechanism of the nose and sinuses. Dwyer [10] asserted that many postoperative infections travel through the lymphoid tissue of the nasopharynx, and Turner and Reynolds [7] offered the rational explanation that infection in the nasopharynx may sometimes travel up through a persistent craniopharyngeal canal (Rathke's) to infect the body of the sphenoid bone and then the meninges.

The well organized exudate at the base of the brain and the thin flocculent purulent exudate that covers the pia in the sulci and on the surface of the brain represent the point of entrance of the infection and nature's effort to confine it.[11] It is the signpost pointing to the field which is to be considered. This area lies over the middle ear, the mastoid, the petrous pyramid, the ethmoid cells, the sphenoid sinus and the basilar process of the sphenoid bone.

The physiologic action of the pia-arachnoid produces a barrier more or less temporary, depending on the virulence of the infection, to the further extension of the disease process. Eagleton [2] pointed out that "this framework is not only a barrier but also contains a protective mechanism, so that any infection which may pass the barrier of the dural covering, or which may be brought to the brain by the circulating blood, will be instantly counteracted, digested and removed." He stated further that "if we are to understand meningitis, we must understand this framework, this barrier, this protective mechanism, this phagocytic membrane, not only in its gross anatomy, but also in its microscopic physiology, and especially its reaction in the presence of an irritant, old trauma, cold, or microorganisms."

SYMPTOMS

Generally the diagnosis of meningitis is not difficult because the clinical picture develops fairly rapidly and is rather impressive. However, when the type III pneumococcus is the invading organism its development may be, and often is, insidious. Bradbeer [12] reported a case of meningitis complicating mastoiditis due to Pneumococcus type

10. Dwyer, J. G.: Present Status of Otitic Meningitis, Laryngoscope **46**:245-251 (April) 1936.

11. Smith, F.: Basal Meningitis, J. A. M. A. **107**:189-194 (July 18) 1936.

12. Bradbeer, W. H.: Case of Meningitis Complicating Mastoiditis Due to Pneumococcus Type III, J. Laryng. & Otol. **51**:241-242 (April) 1936.

III which illustrates its insidious and dangerous nature. Kopetzky [13] stated that too often the spinal fluid gives a positive reaction only at a time when the clinical picture is clearly manifest, and usually too late to be of therapeutic significance. He emphasized [14] the importance of recognizing the condition when the invasion is taking place and of instituting therapy before an inflammatory reaction has taken place and infection begins. An early diagnosis, he said, can be made by a thorough study of the spinal fluid and the blood. Brunner,[8] however, stated that sometimes such a diagnosis is difficult, particularly when there are few clinical signs.

Clinically, pneumococcic infection may present a variable progress. Maybaum and Druss [15] noted (1) a prolonged clinical course with few or no physical signs, followed by evidence of mastoiditis or intracranial complications; (2) the usual clinical course of middle ear infection, followed by mastoiditis or intracranial complications, and (3) a short clinical course (two to five days), followed by intracranial complications.

The symptoms are influenced largely by whether actual systemic involvement, limited meningeal irritation or increased intracranial pressure predominates. The disease is frequently ushered in by a feeling of malaise. Headaches are common and are dull and persistent. Postauricular pain is moderate and persistent in cases of otitic origin, and there may be a dull, heavy sensation between the eyes in the cases of rhinogenic infection. Transitory severe neuralgias are common, and various palsies of the cranial nerves and labyrinthine disturbances may appear. When a discharge from the nose or ears has been present it frequently diminishes, although occasionally it may increase in quantity. Impaired hearing and tinnitus are often noted and may be severe, out of all proportion to the apparently mild involvement of the drum membrane. In 3 cases in this series the drum membrane appeared normal in every respect and yet the mastoid bone was completely destroyed. Persky,[16] in discussing pneumococcic otitic infections, stated that the symptoms indicate a slow progressive otitic infection. Vision may be impaired and examination of the eyegrounds show evidence of intracranial pressure. The patient may or may not complain of pain in the neck but does not have stiffness of the neck until the cisterna magna becomes involved.[11] As the disease progresses, the patient lies on his

13. Kopetzky, S. J.: Diagnosis and Treatment of Otogenic Meningitis, Am. J. Surg. **42**:131-141 (Oct.) 1938.

14. Kopetzky, S. J.: Clinical Picture of Bacterial Meningitis with Particular Reference to Its Changing Phases, Laryngoscope **47**:301-303 (May) 1937.

15. Maybaum, J. L., and Druss, J. G.: Otitic Infections Due to Pneumococcus Type III, Arch. Otolaryng. **30**:21-37 (July) 1939.

16. Persky, A. H.: Pneumococcus Type III in Otitic Infections, Arch. Otolaryng. **34**:473-484 (Sept.) 1941.

TABLE 5.—*Interval Between Onset of Symptoms Referable to Ear, Nose or Throat and Development of Meningitis*

Organism	Interval	
Pneumococcus	Number of Cases	Period
*I	1	3 days
III	11	14 days of discharge from ear; 1 day of headache 7 days left ear; 1 day right ear; no discharge 14 days 10 days right side; 6 days left side 14 days of discharge 21 days of discharge ?; earache treated in clinic 21 days of discharge from right ear 42 days (6 weeks) 3 days after injury 10 days after injury
IV	4	1 day 4 days 8 days 3 days
V	2	3 days ?; arrival in coma
VI	2	Several days ?; general failure of 3 month child to gain weight, while twin gained
VII	1	1 day
VIII	3	1 day 1 day 3 days after operation
X	1	4 days
XII	1	1 day
XIV	1	8 days of diarrhea
?XVII	1	Discharge from ear for three weeks, cessation for three weeks; then fever and three days later meningitis
XVIII	1	7 days
XIX	1	10 days
XX	3	7 days of common cold; 1 day of earache 8 days of pain and discharge from right ear 3 days
XXIII	2	?; diabetes; patient without insulin for 4 days; coma on admission 4 days
XXV	1	1 day
Undetermined	2	8 days ?; chronic infection of middle ear
Streptococcus		
Gamma	2	10 days with infection of upper respiratory tract 5 days
Beta	5	? 12 days 14 days 9 days 5 days
B. Friedländer A	1	50 days
B. xerosis	1	14 days
B. pyocyaneus	1	1 month
H. influenzae	3	8 days 1 day 1 day
B. proteus	1	7 days
Meningococcus	4	3 days 2 days 7 days 7 days
Undetermined	1	?
Trauma; no organisms	1	4 days

back with his eyes closed, in a state of semicoma, from which he is easily aroused; the elevation of temperature is moderate and the pulse relatively slow. The abdominal reflexes may be diminished, and there is a suggestive Kernig sign. At this time there is only moderate leukocytosis. The spinal fluid is under slightly increased pressure at this stage and is usually cloudy, with an increased cell count, but may not show any bacteria. Chemical analysis of the blood and the spinal fluid may present a clue; the findings will be discussed in detail later.

Extraotitic meningeal syndromes must be differentiated; as described by Friedman,[17] they include tuberculous meningitis; meningococcic meningitis; meningismus; serous meningitis; otitic hydrocephalus; cerebral poliomyelitis; encephalitis; subarachnoid hemorrhage; syphilitic meningitis; sympathetic meningitis; influenzal meningitis; torula meningitis; actinomycosis; meningitis produced by trauma, encephalographic examination, the intraspinal introduction of serum or induction of spinal anesthesia; suppurative epimeningitis; subdural hematoma; benign lymphocytic meningitis; primary sarcomatosis, and secondary carcinomatosis of the meninges.

The Levinson test is helpful in determining tuberculous infection of the spinal fluid. In each of two small tubes (0.3 cm. in diameter) 1 cc. of spinal fluid is placed. Into one tube is introduced 1 cc. of 3 per cent sulfosalicylic acid; in the other, 1 cc. of 1 per cent mercury bichloride. The tubes are stoppered tightly, and the immediate precipitation is disregarded. They are allowed to stand for twenty-four hours. Then the height of the precipitate in the test tubes is measured. If the precipitate in the mercury bichloride is three times as high as that in the sulfosalicylic acid, the result is positive for tuberculosis. Otherwise, the results in the two tubes are the same or reversed.

DIAGNOSIS

Of all the laboratory tests at one's disposal, examination of the spinal fluid is the most important. The type of meningitis present may be suspected from the appearance of the spinal fluid. On this point Dwyer[18] had this to say: If there are signs of meningitis and the spinal fluid stays clear, the infection is either tuberculous or syphilitic. If it is tuberculous, then by long centrifugation one can usually demonstrate the tubercle bacilli; if it is syphilitic the cell count is lymphocytic and the serum gives a positive Wassermann or Kahn reaction. Occasionally benign lymphocytic meningitis may occur in conjunction with otitic

17. Friedman, E. D.: Extraotitic Meningeal Syndromes (Differential Diagnosis), New York State J. Med. **38**:335-338 (March 1) 1938.

18. Dwyer, J. G.: Differential Diagnosis of Otitic Meningitis, Laryngoscope **49**:21-22 (Jan.) 1939; footnote 10.

infection,[19] but this commonly shows also a few large mononuclear and polymorphonuclear cells. A cloudy fluid in itself does not mean generalized meningitis. In examining the spinal fluid one must consider the physical characteristics and the chemical, cytologic, serologic and bacteriologic findings. Ten cubic centimeters of fluid, or even less, is sufficient for the performance of all the most important tests on the spinal fluid. Kopetzky [13] suggested that a specimen of the patient's blood be taken just after the spinal tap is finished and both sets of specimens covered with a layer of melted paraffin (of low melting point) to prevent evaporation and other changes. The care of the spinal fluid, particularly the first purulent fluid, is to be emphasized, for if improperly handled it may not be possible to identify the causative organism until it is too late to institute proper specific therapy. While the infecting organism is usually found later with ease, in the early stages the pneumococcus, streptococcus, meningococcus and Bacillus influenzae are sometimes difficult to demonstrate, especially if the patient has already received some form of chemotherapy with bacteriostatic effect. The pneumococcus is easily autolyzed on standing or on incubation. Other organisms have special growth requirements; for example, the meningococcus requires increased carbon dioxide tension for optimal growth.

Cunning [20] stated that in several of his cases, during the course of the disease, the fluid was sterile for days and then unexpectedly organisms appeared.

Eggston,[21] in discussing the difficulties of laboratory diagnosis and the value of cultures, stated that in many cases a report on the type of infection in the spinal fluid and a practical diagnosis can be made in the time it takes to make a Gram stain or a hanging drop study but that these diagnoses are of approximate value only. Any type of bacteria may vary at times in its reaction to the Gram stain. This is particularly true of the meningococcus, the influenza bacillus, the streptococcus and the pneumococcus. Also, the Gram stain and technic vary in different laboratories. It requires judgment and vast experience to give anything but an approximately accurate opinion on the basis of the stained film alone. A culture made from loop implantation may fail to show organisms and culture of a centrifuged specimen may be successful.

Brunner [8] stated that in some cases observed no bacteria could be found in the spinal fluid up to the time of the patient's death, and in other cases there were organisms but no marked increase in cells. In

19. Ersner, M. S.: The Diagnosis of Otitic Surgical Lesions, in Kopetzky, S. J.: Surgery of the Ear, New York, Thomas Nelson & Sons, 1939, p. 73.

20. Cunning, D. S.: Treatment of Otitic Meningitis, Tr. Sect. Laryng., Otol. & Rhin., A. M. A., 1939, pp. 122-148.

21. Eggston, A. A.: Bacteriology of Suppurative Meningitis, Laryngoscope 49:16-20 (Jan.) 1939.

spite of these observations meningitis was discovered at autopsy. However, in the majority of cases the fluid was cloudy and organisms were present.

By the time acute meningitis is suspected clinically, the spinal fluid has already undergone characteristic changes, namely, increase in pressure, increase in the number of cells (polymorphonuclear leukocytes predominating), increase in the protein content and invasion by the infecting organism, demonstrable both on smear and in culture. The fluid, at first merely cloudy, rapidly becomes more turbid and purulent

TABLE 6.—*Organisms Found on Culture in Cases of Otorhinogenic Meningitis*

Organism	Number of Cases	Material from Mastoid	Material from Pharynx	Spinal Fluid
Pneumococcus type				
I	1	1	..	1
III	11	4	..	9
IV	4	1	1	4
V	2	..	..	2
VI	2	1	2	1
VII	1	..	..	1
VIII	3	1	..	2
X	1	..	1	1
XII	1	..	..	1
XIV	1	..	1	1
XVII	1	..	..	1
XVIII	1	1	..	1
XIX	1	..	..	1
XX	3	1	..	3
XXIII	2	2	..	2
XXV	1	1	..	..
Undetermined	2	1	..	1
Streptococcus				
Gamma	2	2	..	2
Beta	5	2	..	3
B. proteus	1	1	..	1
B. Friedländer A.	1	..	..	1
B. xerosis	1	1	..	..
B. pyocyaneus	1	1	..	1
H. influenzae	3	..	..	3
Meningococcus	4	..	..	4
Undetermined	1	..	..	..
Traumatic; no organisms	1	..	..	..

and contains increasing numbers of organisms and cells and an increasing quantity of protein and fibrin, either preformed or forming in the test tube. The sugar content is reduced early and rapidly falls. It must be emphasized that the test for sugar should be performed immediately after aspiration of the fluid because the sugar disappears in vitro. The chlorides are usually lowered. In serous meningitis there are no bacteria and the sugar content is normal.

In the subacute stage there is a tendency for mucofibrinous masses to accumulate in different loci of the spinal and cerebral subarachnoid spaces, especially in the lumbar sac and in the upper cervical portion of the spinal canal. Under such conditions the fluid is thick, purulent and scanty. The organisms may be numerous or may have been destroyed. In these cases puncture must be made at a higher level, and it may not be

amiss to describe in detail the technic of making a cisternal puncture. The hair on the back of the neck is shaved to the external occipital protuberance. The area in the midline from this point to the second cervical spine is painted with iodine. The patient is placed in a position so that his head is on a straight line with the cervical portion of his spine. The neck is flexed anteriorly about 20 degrees. The thumb of the left hand palpates and finds the midline in the area between the external occipital protuberance and the spine of the second vertebra. The needle is inserted inward at an angle so that it points in a line between the external auditory meatus and the glabella. It should never be inserted more than 6 cm. It passes through the subcutaneous tissue and the muscles and strikes the firm, resistant occipitoatlantoid ligament. It takes considerable pressure to pass through this portion of the dura, and care must be exerted that the needle is not pushed too far anteriorly by the force that is required to penetrate the dura. Difficulties in obtaining fluid are usually due to being outside the midline or to not directing the needle in the proper direction. Anomalous veins sometimes give rise to bleeding, and when this occurs no further puncture should be made. There is some danger in this procedure, and deaths have been described which were actually due to the cisternal puncture, whereas lumbar puncture is entirely free from danger when contraindications to withdrawal of fluid do not exist.

Kopetzky [13] emphasized that it is in the chemical study of the fluid that the most important diagnostic factors are obtainable. The exact estimation of the carbohydrate present in the spinal fluid in a successive series of taps gives an indication of the development of bacterial infection. The observation of bacteremia is of diagnostic significance. Estimation of the chlorides, the carbon dioxide, the lactic acid and the p_H is also made, and the results are compared with those obtained in a similar study on the blood. In meningitis lactic acid is much increased in the fluid as compared with the blood. Any patient with a continuing high fever will have an increase of lactic acid in the blood, but in meningitis the disproportion of lactic acid in the spinal fluid to that in the blood is 3 or 4 to 1. Normally there are more chlorides in the spinal fluid than in the blood. In meningitis the amounts become more nearly equal. There is a marked decrease of the bicarbonate content of the spinal fluid, often as low as 33 per cent below that of the blood plasma.

The p_H of the spinal fluid in meningitis is much lower than normal. In this disease it is almost always lower than that of the blood. The normal spinal fluid is practically protein free, the protein nitrogen ranging from 35 to 100 mg. per hundred cubic centimeters, while the protein content of the plasma ranges from 6,500 to 8,500 mg. per hundred cubic centimeters. The clinician must remember that for all practical

purposes the ratio of lactic acid in the fluid to that in the blood is normally 1 to 1. In meningitis the ratio of lactic acid in the fluid to that in the blood is 3 to 1. There is, therefore, a drop in the chlorides, the carbon dioxide tension and the p_H.

Determination of the chloride content and the finding of an increase in lactic acid, lowering of the protein content and loss of reducing substances may be important adjuncts to diagnosis, but the presence or absence of an infecting organism, the increase or decrease in the cell count and the spinal fluid pressure are the cardinal factors used to determine the pathologic process and the patient's status. We therefore have subjected the spinal fluid only to routine serologic tests and a quantitative estimation of sugar except in cases in which special indications were present.

MECHANISM OF PRODUCTION OF MENINGITIS

All meningitides are generalized and similar but exhibit different phases. The meningeal picture is constantly changing because one is dealing with a circulating fluid—a pulsating, moving, semisolid mass which supports the whole cerebrospinal system. The cerebrospinal fluid is secreted by the choroid plexus. It is absorbed by the pacchionian bodies, through which it reaches the general circulation. The brain and the fluid are not compressible; therefore the blood must exert more pressure than the cerebrospinal fluid to get through the cell walls. As a result of the increased blood pressure, more cerebrospinal fluid is secreted by the choroid plexus; this of course raises the cerebrospinal fluid pressure. Thus a vicious cycle is produced, so that the blood pressure is constantly being increased until the respiratory centers become paralyzed.

Symptoms of toxicity may predominate when the products of cerebral metabolism are not removed by a freely circulating cerebrospinal fluid. Bacteria entering the central nervous system through the blood live on dextrose and are fermentative; their activity, therefore, is shown by progressive loss of carbohydrate. When the sugar is exhausted they attack the protein, producing putrefaction. Bacteria are usually not found in the spinal fluid culture while there is still sugar, one or two days intervening between the disappearance of sugar and the appearance of bacteria. As a result of the putrefaction, the tissue of the brain degenerates into choline and marsh gas products, such as ammonia and ammonia products. The intracranial pressure increases and compresses the blood vessels, so that proper oxygenation is interfered with. The lessened alkalinity resulting from improper oxygenation produces a colloidal edema, which increases in turn the intracranial edema, with resultant collapse of the veins and the arteries. These vessels are unable

then to remove the waste products. Choline appears in excess in the spinal fluid, and the patient dies of choline poisoning and toxemia.

TREATMENT

Historical Condensation.—Up to the advent of sulfanilamide and its derivatives the mortality of meningitis, particularly of the otitic type, when bacteria were present in the spinal fluid was almost 100 per cent. In fact, Ersner stated that in twenty-five years of otologic practice, until he began to use sulfanilamide, he saw recovery in only 2 cases. All kinds of treatment had heretofore been tried. Among the somewhat unusual cases is that reported by Hamon and Bolzinger [22] of a patient who was treated with Vincent's serum and recovered. However, since the report speaks of the spinal fluid being clear and the culture sterile, it is perhaps injudicious to attribute the cure to administration of the serum, especially in view of the fact that mastoidectomy was done in this case and it has been established that such an operation performed before the appearance of organisms in the spinal fluid frequently produces a cure.

Adam and Connal [23] reported 9 consecutive cases, with 7 recoveries. They opened the dura without exception in these cases. However, a close study of the reports shows that in case 1 there was no growth on culture of the spinal fluid, in case 2 only a few gram-positive diplococci were revealed in the first puncture while later punctures showed no organisms, in case 4 the fluid was sterile, in case 5 a few degenerated diplococci were obtained on first puncture and later punctures yielded no organisms and in case 6 no organisms were obtained on smear or in culture but later cocci appeared in the smears and the patient died. In case 7 streptococci were observed in the fluid and "prontosil" (form not given) was administered intramuscularly. This was the first case of the series in which the drug was given. In case 8, in which the patient died, and in case 9 no organisms were obtained on puncture but were present in the fluid on incision of the dura. In summarizing, the authors stated that those who regard a positive culture essential for diagnosis would dispute the inclusion of some of these cases.

Scott and Radbill [24] reported a case of meningitis due to Streptococcus haemolyticus in which recovery followed mastoidectomy and the

22. Hamon and Bolzinger: Méningite à streptocoques hémolytiques d'origine otitique, traitée par le serum de H. Vincent et la mastoïdectomie. Guérison, Bull. Acad. de méd., Paris **115**:163-166 (Jan. 21) 1936.

23. Adam, J., and Connal, E. A. M.: Purulent Meningitis: Nine Consecutive Cases with Seven Recoveries, J. Laryng. & Otol. **52**:87-97 (Feb.) 1937.

24. Scott, J. P., and Radbill, S. X.: Streptococcus Hemolyticus Meningitis with Recovery Following Mastoidectomy and Intrathecal Administration of Lyophile Convalescent Scarlet Fever Serum, J. Pediat. **10**:486-489 (April) 1937.

intrathecal administration of lyophile convalescent scarlet fever serum. They added that improvement began before administration of the convalescent scarlet fever serum was started but that some facts made it possible that the operation alone was not responsible for the child's recovery. In our opinion this is pure speculation, particularly since improvement had already begun before the serum was given.

Shambaugh [25] cited an article by Ferris Smith calling attention to the basilar meningitis which is the beginning of the generalized meningitis of otitic or nasal origin and advocating early incision and drainage of the dura beneath the pontile cistern by way of a burr opening in the sphenoid bone, made through its posterior wall or through the roof of the nasopharynx. He reported 3 cases, with 1 recovery. (In our opinion this procedure in the hands of most operators will do more harm than good. Even in the hands of Ferris Smith there was recovery in only 1 of 3 cases.) Shambaugh then stated that the following principles are coming to be generally accepted: Lumbar puncture should be done immediately at the beginning of meningitis; if the cell count is elevated but there are no organisms on the smear, the focus (sinus, labyrinth or mastoid) should be drained. In the mastoidectomy, the dura should be exposed wide but not incised. If the meningitis is generalized and the smear and culture are positive, surgical drainage of the basilar cistern should be carried out by the Eagleton method,[26] or perhaps by the method of Ferris Smith, which is open to the theoretic objection of exposing the spinal fluid to the mixed flora of the nasopharynx. As long as the meningitis is localized only a thorough surgical drainage of the focus in the ear or sinus should be instituted. Once it is generalized, repeated lumbar punctures and the intracarotid administration of antiseptics may be employed, or incision and drainage of the dura may be considered and forced drainage of the dura by the method of Kubie instituted. In this article there is still no mention of sulfanilamide and its derivatives.

Kubie's method of forced drainage of the spinal fluid consists in a combination of continuous drainage and the intravenous administration of hypotonic solution of sodium chloride, half physiologic strength (0.45 per cent). Lumbar puncture is done, and the needle is allowed to remain in situ. At first it was allowed to remain in place for eight hours at a time; later this was reduced to only three hours. Two thousand cc. of hypotonic solution of sodium chloride is given intravenously. Both these procedures were carried out every day. The whole method has

25. Shambaugh, G. E., Jr.: Surgical Treatment of Meningitis of Otitic and Nasal Origin, J. A. M. A. **108**:696-700 (Feb. 27) 1937.

26. Eagleton, W. P.: Otitic Meningitis, J. A. M. A. **87**:1544-1548 (Nov. 6) 1926.

now been almost completely discarded, as experience has shown no beneficial results that could be ascribed to it. Many workers even feel that it may occasionally produce a harmful effect, because the irritation of the needle may contribute to the production of a spinal block.

Jobson [27] stated, "Slight progress has been made in solving the problem of otitic meningitis as compared to other otological conditions." He made no mention of sulfanilamide and its derivatives.

Neal, Jackson and Appelbaum [28] classified attempts at treatment under three heads: (1) surgical procedures, (2) serologic and allied therapy and (3) chemotherapy. The more radical surgical methods of establishing drainage, such as laminectomy or trephining the cisterna magna, have no advantage over repeated lumbar punctures. The authors do not recommend forced spinal or perivascular drainage. Treatment of forms other than meningococcic meningitis with serums gave disappointing results. In treatment of pneumococcic meningitis they used specific serum frequently, without a single recovery. In management of streptococcic meningitis they used antistreptococcus serum, with occasional recoveries, but they expressed the belief that recovery was not due to the serum. In 2 cases, however, in which the meningitis followed scarlet fever complicated by otitis media they claimed that recovery was due to the specific action of the antibacterial serum. Chemical agents, they said, are of doubtful value and occasionally do harm. Up to this point there is no mention of sulfanilamide. The article continues, however, with the following statement:

> In recent months the use of para-aminobenzenesulphonamide has received widespread attention . . . It has been shown by work with animals that it is highly effective in its action on the hemolytic streptoccocus, the meningococcus and the pneumococcus. Its effect on other organisms appears doubtful . . . It may act in infections with the influenza bacillus. . . . Clinical studies at present do not seem to confirm the results of animal experimentations with the pneumococcus. The mode of action of this chemical is not understood. . . .
>
> We have used Prontosil and sulfanilamide usually in the form of Prontylin in cases of meningitis due to the hemolytic streptococcus, the pneumococcus and the influenza bacillus. We have also used this chemical in a few cases of meningococcic meningitis. . . .
>
> We have treated 9 cases of pneumococcic meningitis. At present only one has recovered. Seven have died and one is still under treatment.
>
> Two cases of influenza meningitis have received this chemical agent. . . . with one recovery.
>
> The use of Prontosil and Prontylin in cases of hemolytic streptococcus meningitis have yielded results that seem to us quite astounding. . . . We have had

27. Jobson, G. B.: Otitic Meningitis, Pennsylvania M. J. **40**:735-739 (June) 1937.

28. Neal, J. B.; Jackson, H. W., and Appelbaum, E.: Summary of Methods Used in Treating Meningitis Secondary to Infections of Ears and Sinuses, Laryngoscope **47**:317-322 (May) 1937.

twelve cases. In every instance the diagnosis was made by culturing the hemolytic streptococcus from the spinal fluid. All but one have been secondary to infections of the ears and mastoid; this patient had a pansinusitis. Nine have recovered, two have died, and the twelfth is still under observation. [One] died within 12 hours . . . and the [other] within 24 hours after the first dose of prontosil.

The article continues:

During . . . 1936 we saw 20 cases of hemolytic streptococcic meningitis, all of which died with the exception of one following scarlet fever.

As to the mode of administration, they stated:

Since it has been shown by Marshall, Emerson and Cutting that soon after oral administration the concentration of sulfanilamide in the spinal fluid is nearly equal to that of the blood, intraspinal injections [are] unnecessary.

Bratesco and Racoveano [29] simply recommended surgical intervention because it will afford a greater number of cures but did not give sufficient statistics on the results and did not mention the use of sulfanilamides.

Wasowski [30] reported 3 cases in which favorable results followed intraspinal insufflation of air. This is too small a number from which to draw conclusions. There is no mention of drugs of the sulfanilamide group.

Adam and Connal [31] reported 2 cases, with recovery in 1. No drugs of the sulfanilamide group were used.

Kopetzky [14] recommended removal of the focus and a small blood transfusion repeated every second day, especially during the stage of invasion.

Kline [32] reported 2 cases of recovery of otitic meningitis. He mentioned Caussé, Loiseau and Gisselbrecht [33] as the first to report such a recovery after treatment with original prontosil (the hydrochloride of 4-sulfamido-2′,4′-diaminoazobenzene). He expressed the belief that there is no justification for the radical methods of spinal drainage. While he stated that incision of the dura facilitates drainage, this was not done in the cases he reported. He expressed the opinion that serum and sulfanilamide are the two most important therapeutic agents.

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29. Bratesco, V., and Racoveano, V.: Sur le traitement chirurgical de la méningite otogène, Ztschr. f. Hals-, Nasen- u. Ohrenh. **40**:657-660, 1937.

30. Wasowski, T.: Ueber die Behandlung der otogenen eitrigen Hirnhautentzündung mittels intraspinaler Lufteinführung, Monatschr. f. Ohrenh. **71**:557-561 (May) 1937.

31. Adam, J., and Connal, E. A. M.: Two Cases of Otitic Meningitis with Recovery in One, J. Laryng. & Otol. **52**:497-500 (July) 1937.

32. Kline, O. R.: Meningitis of Otitic Origin: Report of Two Cases, Arch. Otolaryng. **37**:739-745 (June) 1938.

33. Caussé, Loiseau and Gisselbrecht: Méningite purulente otogène à streptocoques hémolytiques, traitée exclusivement par un colorant azoïque. Guérison, Ann. d'oto-laryng. **2**:194-199 (Feb.) 1936.

Appelbaum [34] said that experimental work indicates that a combination of azosulfamide and specific serum has some curative action on otherwise fatal inoculations of mice with influenza bacilli. With the same treatment for patients the results have been disappointing. Most of his patients who recovered were not treated by intraspinal administration of sulfanilamide, and he repeated what he had already said at other times, that he did not recommend forced spinal or perivascular drainage, because adequate drainage is accomplished by repeated spinal punctures, or by cisternal or ventricular puncture if a block occurs.

This was also the stand of Aries,[35] who asserted that continuous spinal drainage simultaneous with the intravenous administration of a hypotonic saline solution has been advised by Kubie and Reton but that he has seen no beneficial results in this type of treatment.

Prognosis.—Before the additional use of the sulfanilamide drugs the mortality, particularly that of otitic bacterial meningitis, was practically 100 per cent.

Dwyer [10] stated:

> In 28 years I have seen 367 cases of meningitis with 18 recoveries, and why they recovered I do not know. . . . I do not believe it was anything we did that cured them.

The following year he stated that the use of sulfanilamide alone or combined with allied substances was a new development. "The future looks a little brighter for the solution of this problem."

Among the first to mention the drugs of the sulfanilamide group were Lewy and Hagens,[36] who stated:

> Streptococcus hemolyticus (especially the beta strain) seems to offer more hope than before the use of . . . prontosil. Any pneumococcus meningitis is practically hopeless at present.

Surgical Treatment.—The moment the diagnosis of meningitis is suspected, a thorough search must be made for an apparent focus of infection of otorhinogenic origin. If such a search for a focal source fails to show at least some basis for suspecting the ears or sinuses, then no operation should be performed, for the meningitis may be secondary to pneumonia or some other medical condition, in which case the additional physical drain of an unnecessary operation might contribute to a fatal outcome. But there should be no delay in eliminating surgically

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34. Appelbaum, E.: Symposium on Otitic Meningitis: Chemotherapy in Meningitis Secondary to Ear and Sinus Affections, Laryngoscope **49**:30-45 (Jan.) 1939.

35. Aries, P. L.: Pediatric Treatment of Otologic Sepsis Including Meningitis, Illinois M. J. **75**:131-136 (Feb.) 1939.

36. Lewy, A., and Hagens, E. W.: Report of Chicago Committee on Otitic Meningitis, Laryngoscope **47**:761-775 (Oct.) 1937.

TABLE 7.—*Incidence of Involvement of Sinuses and Ears*

Organism	Number of Cases	Sinuses	Mastoid			Appearance of Drum Membrane
			Left	Right	Both	
Pneumococcus	1				1	Both bulging; yellow pus revealed on myringotomy
III	11				1	Bilateral discharge
					1	Left bulging; right injected
				1		Pus; discharge
			1			Perforation; bulging; pus
		Maxillary	1			Profuse discharge
		Pansinusitis on roentgenogram				Normal
			1			Oily discharge; red and bulging
				1		Red and bulging; pinpoint perforation
			1			Drums not visualized
		Maxillary				Normal
		Frontal				Normal
IV	4		1	1		Bleeding from middle ear
						Normal
		Frontal				Normal
		Nasal secretion and cloudy antrum				Both retracted, with little light reflex
V	2		1			Bulging and red
				1		Injected
VI	2		1			Bulging
				1		No examination of ears, nose and throat
VII	1				1	Pulsating; discharge; bulging
VIII	3		1			Red and bulging
			1			Recent myringotomy, with discharge
		Sphenoid and ethmoid				Normal
X	1	Sphenoid				Normal
XII	1				1	Injected
XIV	1	Positive smear and culture from pharynx				Normal
XVII	1				1	Dull, thick, scarred
XVIII	1				1	Gray; no bulging
XIX	1	Sinusitis				Normal

XX	3		1		Greenish discharge
				1	Purulent discharge
		Frontal			Normal
XXIII	2		1	1	Laceration and blood
					Left bulging
XXV	1	Right maxillary and ethmoid			Normal
					Injected; bulging; pus
Undetermined	2			1	Perforation and pus
			1		Gray and bulging
Streptococcus					
Gamma	2			1	Gray; pulsating; discharge
Beta	5	Sphenoid		1	Perforation of right drum
				1	Otorrhea; pus; pulsating under pressure
				1	Bilateral discharge
		Right antrum			Normal
		Frontal; ethmoid		1	Bleeding
B. Friedländer A	1			1	Perforated bilaterally; dull; gray
B. xerosis	1		1		Profuse pulsating discharge; central perforation
B. pyocyaneus	1		1		Bulging with purulent discharge
H. influenzae	3	Sphenoid and ethmoid			Normal
		Sphenoid and ethmoid			Normal
		Pharynx red; positive culture from nose and throat			Normal
B. proteus	1		1		Bulging
Meningococcus	4	Sinusitis			Normal
		Frontal			Normal
		Cold and sore throat			Normal
		Injected pharynx		1	Red; bulging
Undetermined	1		1		Normal
Traumatic; no organisms	1		1		Normal

Abnormal drums with sinusitis	1	Drum involved but not bulging	6
Normal drums with sinusitis	18	Drums not visualized	2
Normal drums with mastoiditis	3	Bleeding from middle ear	3
Bulging drums without discharge	11		
Bulging drums with discharge	9	Total	58
Discharge from perforation without bulging	5		

a focal source if there is any reason to suspect its existence. Cawthorne [37] cited Eggston, who showed that in 90 of 124 cases of fatal meningitis encountered over an eight year period (up to 1934) the cause was aural disease. He continued: "Thus we see that the ear is the commonest cause of septic meningitis, which is the most frequent cause of death in disease of the ears, nose and throat."

Frequently it is difficult to determine whether the sinuses or ears are at fault and on which side of the head the apparent focus is situated. Even after a careful clinical and roentgenographic examination, one may be at a loss to know on which side to operate. It is in these cases of vague localization that a detailed history will be of great value. We have encountered cases of meningitis in which the drum membranes appeared absolutely normal and we were induced to operate because there was no other apparent cause for the meningitis and because some member of the family was able to state definitely that at some time previous there had been a discharge from the ear. In these cases surgical intervention revealed extensive destruction, as shown in table 7.

In cases of otitic meningitis a definite pathologic condition is often revealed in the middle ear, the mastoid or the petrous pyramid, but it is rare to be able to demonstrate macroscopically a direct pathway of infection through erosion. Therefore in operating on the mastoid one is breaking up the continuity of the infection from the diseased area along the perforating vessels going to the dura—hence the rationale of taking down extensively the dural plate of the mastoid and a good portion of the inner table. This decompresses the brain and allows better circulation, with the consequent removal of waste products. Some workers, notably Cunning,[20] have advocated making multiple parallel incisions in the dura to allow cerebrospinal fluid drainage. But there is a good deal of disagreement on this point, as spinal fluid sufficient to decompress the brain can be removed by repeated lumbar punctures except in cases of spinal block, which is rather an unusual development. In not 1 of our entire series of cases did this condition develop. We have not thought it advisable to incise the dura in any instance. In cases, now rare, of suppurative labyrinthitis, the labyrinth should be opened, and when the petrous pyramid is involved, as shown roentgenographically and clinically, it should be opened, preferably by the "unlocking" operation of Eagleton.

In cases of rhinogenic meningitis we are in accord with Kramer and Som [9] as to the necessity of removing the diseased mucosa in the accessory sinuses, especially in the posterior ethmoid and the sphenoid. Incidentally, these authors stated that most of their cases represented an acute exacerbation of a chronic submucosal abscess, a statement which

37. Cawthorne, T.: Otogenic Meningitis, J. Laryng. & Otol. **54**:444-469 (Aug.) 1939.

is at variance with the older dictum expressed by many investigators, namely, that otogenic meningitis most frequently results from a chronic suppuration of the ear while rhinogenic meningitis most often occurs with

TABLE 8.—*Pathologic Changes Observed at Operation*

Organism	Number of Cases	Operative Observations *
Pneumococcus		
I	1	None
III	11	None
		None
		Free pus and necrotic cells
		Pus and necrotic cells
		Pus and necrosis
		Normal
		Destruction of cells
		Pus and necrosis
		Granulation tissue but no pus
		None
		None
IV	4	Fracture in posterior part of mastoid across middle fossa
		Cells full of blood; dural plate fractured; macerated brain tissue
		None
		None
		Pus in sphenoid sinuses
V	2	None
		None
VI	2	None
		None
VII	1	None
VIII	3	None
		Pus and necrosis
		Sphenoethmoid sinuses full of pus bilaterally
X	1	None
XII	1	None
XIV	1	None
XVII	1	None
XVIII	1	None
XIX	1	None
XX	3	None
		Soft bone and hemorrhagic areas
		None
XXIII	2	None
		None
XXV	1	Pus from antrum
Undetermined	2	None
		Chronic infection
Streptococcus		
Gamma	2	None
		Pus and necrotic bone cells
Beta	5	None
		Broken-down cells and petrositis
		Pus and necrosis; soft base of petrous pyramid
		Compound fracture of maxilla, zygomas and nasal bones
		None
B. Friedländer A	1	Pus and necrotic cells
B. xerosis	1	Cells filled with mucopus
H. influenzae	3	None
		None
		None
B. proteus	1	Foul pus under pressure; perisinal abscess
Meningococcus	4	None
		None
		None
		None
Undetermined	1	Sphenoethmoiditis
Trauma; no organism		Bullet in mastoid

* "None" means that no operation was performed.

an acute infection. Table 5 shows that in our series the rhinogenic forms were acute.

Supplementary Treatment.—Carefully controlled cerebrospinal fluid drainage is a widely used method of combating cerebral edema, although

Hoyne [38] expressed the opinion that it may be harmful. The method should have application in treating the cerebral edema of meningitis. It is believed by some workers that this procedure also removes inflammatory products in helpful quantities and that reestablishment of a normal circulation of the blood and cerebrospinal fluid is promoted. There is the possibility that the periodic removal of cerebrospinal fluid facilitates the transfer of the sulfanilamide drugs through the choroid plexus. The observed increase of these drugs in the cerebrospinal fluid after drainage and the subsequent clinical improvement of some of the patients in this series suggest the value of maintaining a relatively normal level of intracranial pressure by repeated lumbar puncture or wound tapping. The character of the fluid may be such that the drainage needle becomes occluded and a false pressure reading is obtained. In such a case one may be tempted to stop drainage after only a small quantity of fluid has been removed, when considerable amounts remain. It may be advisable and helpful to elevate the head of the bed and compress the jugular veins to increase the drainage.

We advocate the use of repeated small transfusions of blood, 150 to 200 cc. for adults and 75 to 100 cc. for children, the purpose being to stimulate the hematopoietic system and to help in reversing the abnormal spinal fluid status to one as normal as possible.[39] Repeated infusions aid in avoiding dehydration.

Chemotherapy.—Preceding the use of sulfanilamide and its derivatives, in 1937, bacterial meningitis proved to be almost invariably fatal. The year before, Tripoli [40] reported a series of 111 cases, of which 110 terminated fatally. The literature now contains a great many reports of successful cure in cases of this disease.

Spink [41] stated that the primary action of the sulfanilamide drugs is one of bacteriostasis; they reduce the rate of growth of bacterial cells and in some conditions kill a small number by interfering with their normal metabolism. All of the major compounds of this group are effective when administered orally. Though there are differences in the rate of absorption, due largely to the differences in solubility of the compounds, the absorbed drugs diffuse to all tissues and body fluids.

In any instance in which there is delay or doubt on the part of the laboratory in establishing or definitely confirming a bacteriologic

38. Hoyne, A. L.: Therapy of Cook County Hospital: Therapy of Meningitis, J. A. M. A. **117**:1973-1978 (Dec. 6) 1941.

39. Kopetzky, S. J.: Newer Concepts of Otogenic Meningitis, Laryngoscope **45**:827-838 (Nov.) 1935.

40. Tripoli, C. J.: Bacterial Meningitis: A Comparative Study of Various Therapeutic Measurements, J. A. M. A. **106**:171-175 (Jan. 18) 1936.

41. Spink, W. W.: Sulfanilamide and Related Compounds in General Practice, Chicago, The Year Book Publishers, Inc., 1941.

diagnosis, it is generally advisable to begin treatment with sulfadiazine (2-[paraaminobenzenesulfonamido]-pyrimidine), since Long and his colleagues [42] have found it effective in controlling experimental infections due to the pneumococcus, staphylococcus and hemolytic streptococcus and since these are the causative organisms in the largest number of cases of meningitis. Observations in our series corroborate the fact. It is self evident, however, that the intelligent use of sulfanilamide and its derivatives requires precise bacteriologic study.

Doses of the sulfanilamide group of drugs vary with the severity of the infection and with the individual tolerance of the patient. In general, one gives 1 to 1½ grains (0.065 to 0.096 Gm.) per pound (0.5 Kg.) of body weight in twenty-four hours, the initial dose being about one-half the calculated twenty-four hour dose. If the patient cannot tolerate or is too ill to receive oral medication, the drug may be administered parenterally. Sulfanilamide in 1 per cent solution in sodium lactate may be given only subcutaneously or rectally, while the sodium salts of the others may be given intravenously in 5 per cent solution in sterile physiologic solution of sodium chloride or in distilled water, as well as subcutaneously.

As to untoward reactions to chemotherapy, it is well to remember that individual tolerance to the drugs differs. If there is any previous history of drug sensitivity, a small oral test dose may be administered and about twelve hours allowed to elapse. In the absence of toxic manifestations, such as a cutaneous eruption, or of any increase in the symptoms of nausea, fever and headache, which may heretofore have been present, intensive therapy may be instituted. In the order of their deleterious effects on the liver, sulfanilamide seems to be the worst offender, followed by sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine), sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) and sulfadiazine.

Serum.—Specific serum, with the production of antibodies, is still, in our experience, a valuable adjunct in the treatment of meningitis and may be used in combination with chemotherapy. It is employed, of course, in the treatment of meningococcic and of the other bacterial meningitides, particularly in case of those patients who tolerate sulfanilamide and its products poorly and those who show no favorable response to chemotherapy within forty-eight hours. Serum was administered in 25 of our cases. It is also indicated, according to Spink,[41] for persons over 50 years of age, patients with chronic alcoholism and women in the last trimester of pregnancy or in the puerperium.

42. Long, P. H.; Bliss, E. A., and Ott, E.: Studies on Sulfadiazine: Chemotherapy of Experimental Haemolytic Streptococcic, Pnemococcic and Staphylococcic Infections in Mice, Bull. Johns Hopkins Hosp. **69**:297-302 (Oct.) 1941.

TABLE 9.—*Data on Treatment of Otorhinogenic Meningitis*

Pneumococcus Organism	Number of Cases	Drug	Blood Concentration, Mg. per 100 Cc.	Specific Serum, Units	Surgical Treatment	Result
I	1	None	None	None	None	Death
III	11	Sulfanilamide	None	None	None	Death
		Sulfanilamide	None	725,000	None	Death
		Sulfapyridine	8.4	320,000	Mastoidectomy	Recovery
		Sodium sulfapyridine	28.2	425,000	Mastoidectomy	Death
		Sodium sulfapyridine	12.3	None	Mastoidectomy and antrotomy	Death
		Sodium sulfapyridine	None	520,000	Bilateral mastoidectomy, sphenoidectomy, ethmoidectomy and antrectomy	Death
		Sulfapyridine	14.9	674,000	Mastoidectomy	Death
		Sulfadiazine	39.5	360,000	Mastoidectomy	Recovery
		Sulfadiazine	28.2	442,000	Mastoidectomy	Recovery
		Sulfanilamide and sulfapyridine	None	None	None	Death
		Sulfapyridine	None	None	None	Death
IV	4	Sulfanilamide	None	300,000	Mastoidectomy with incision of dura	Death
		Sodium sulfapyridine	27.0	250,000	None	Death
		Sulfanilamide and sulfapyridine	None	None	None	Death
		Sodium sulfapyridine	11.0	390,000	Sphenoethmoidectomy	Death
V	2	None	None	66,000	None	Recovery
		Sodium sulfapyridine	None	?	None	Death
VI	2	Sulfapyridine	None	None	None	Death
		Sulfadiazine	None	None	None	Death
VII	1	Sulfanilamide	None	1,043,000	None	Death
VIII	3	Sulfanilamide	None	6,000	None	Death
		Sodium sulfapyridine	15.3	204,000	Left mastoidectomy	Death
		Sodium sulfapyridine	17.4	None	Bilateral sphenoidectomy	Recovery
X	1	Sulfapyridine	None	740,000	Sphenoethmoidectomy	Death
XII	1	Sulfapyridine	8.5	360,000	None	Death
XIV	1	Sodium sulfapyridine	None	200,000	None	Death
XVII	1	Sulfapyridine	None	160,000	None	Death in 12 hours
XVIII	1	Sulfanilamide	None	20,000 M.*	None	Death
				75,000 Pn.	None	Death
XIX	1	Sulfanilamide	None	740,000	None	Death
XX	3	Sulfapyridine	None	160,000	None	Death in 8 hours
		Sulfapyridine	7.0	294,000	Mastoidectomy	Recovery
		Sulfapyridine	None	224,000	None	Death

* M stands for meningococcus serum.

XXIII	2	Sulfapyridine	None	None	None	Death
		Sulfapyridine	None	None	None	Death
XXV	1	Sodium sulfapyridine	17.3	None	Nasoantral window	Death
Undetermined	2	None	None	None	None	Death
		Sodium sulfapyridine	11.0	None	Mastoidectomy	Recovery
Streptococcus					None	Death
Gamma	2	None	None	None	Right mastoidectomy	Recovery
		Sulfapyridine	None	None	None	Death
Beta	5	Sodium sulfapyridine	None	None	Mastoidectomy	Recovery
		Sulfapyridine	8.4	None	Mastoidectomy	Recovery
		Sulfadiazine	None	None	Repair	Death
		Sulfapyridine	9.3	None	None	Recovery
		Sulfathiazole	0.8	None	Mastoidectomy	Death
B. Friedländer A	1	Sulfapyridine	None	None	Mastoidectomy	Recovery
B. xerosis	1	Sulfapyridine	2.3	None	None	Recovery
B. pyocyaneus	1	Sulfapyridine	None	None	None	Death
H. influenzae	3	Sulfanilamide	None	None	None	Death
		Sulfanilamide	None	None	None	Death
		Sulfapyridine	11.15	None	Mastoidectomy	Death
B. proteus	1	Sodium sulfapyridine	16.0	None	None	Recovery
Meningococcus	4	Sulfanilamide	None	60,000 (250 Cc.)	None	Recovery
		Sulfanilamide	None	300,000	None	Death
		Sodium sulfapyridine	None	None	None	Recovery
		Sulfadiazine	17.6	None	Ethmoidectomy	Death
Undetermined	1	Sulfanilamide	None	None	Mastoidectomy	Recovery
Traumatic, no organisms	1	Sulfapyridine	10.0	None		

Chemotherapy	Number of Cases
Sulfanilamide	12
Sulfapyridine	21
Sodium sulfapyridine	13
Sulfathiazole	1
Sulfadiazine	5
None	4
Sulfanilamide and sulfapyridine	2
Total	58

		Died	Recovered
Received serum	25	18	7
Received no serum	33	22	11
Chemotherapy	54	37	17
No chemotherapy	4	3	1
Surgical treatment	24	13	11
No surgical treatment	34	27	7
Chemotherapy and operation without serum	13	6	7
Serum and operation without chemotherapy	None	....	....
Chemotherapy and serum without operation	13	11	2
Chemotherapy and serum with operation	11	7	4

PNEUMOCOCCIC MENINGITIS

Pneumococcus type III produces a "progressive, insidious type of infection." It is more likely to affect well pneumatized bone and has a distinct tendency to involve intracranial structures. Persky,[16] in discussing pneumococcic infections, stated that the symptoms indicate a slow progressive otitic infection; the discharge is persistent, usually thin and watery, but may be mucoid or mucopurulent and moderate in amount. There does not seem to be any appreciable increase in quantity, and this invites a dangerous "watchful waiting" attitude. The disease usually occurs in older patients. Postauricular pain is moderate, dull and persistent. Headaches are common. The elevation in temperature is moderate. Roentgenograms show extensive pneumatization. Impaired hearing and tinnitus are frequently noted. The tinnitus, deafness and, sometimes, pain are severe and out of all proportion to the appearance of the drum membrane, which, perhaps, shows only slight involvement.

It is obvious, then, that it is frequently difficult to evaluate the findings and to determine the proper time to operate. We are now inclined to open the mastoid or sinuses as soon as the diagnosis of meningitis is established and there is an apparent focus in these areas. When the focus is not apparent, but only suspected, chemotherapy should be given a reasonable trial. There being no remission in the symptoms or signs of meningitis, the suspected focus should be eradicated.

If possible, the type of Pneumococcus in the spinal fluid should be determined before chemotherapy is begun, because the sulfanilamide drugs may alter the type of reaction and incidentally obscure the roentgenographic interpretation. Sulfadiazine and sulfapyridine are indicated, the former being preferred. A blood concentration of the drug of 15 mg. per hundred cubic centimeters is ideal for maintaining the optimum spinal fluid level of 10 mg. per hundred cubic centimeters. As table 9 shows, we have reached a level as high as 39 mg. per hundred cubic centimeters with sulfadiazine and 27 mg. with sodium sulfapyridine.

When serum is to be used, and generally such a decision is advisable, the specific type rabbit serum in average doses of from 250,000 to 725,000 U. S. P. units is administered intravenously. Lately several patients with pneumococcic meningitis have been treated successfully without the use of specific serum.[43] We have only 1 case in which the patient recovered without receiving serum. Eventually it may be found that chemotherapy without serum is as effective as the combined treatment in most cases of pneumococcic meningitis.

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43. Plummer, N.; Liebman, J.; Solomon, S.; Kammerer, W. H.; Kalkstein, M., and Ensworth, H. K.: Chemotherapy Versus Combined Chemotherapy and Serum Treatment of Pneumonia, J. A. M. A. **116**:2366-2371 (May 24) 1941.

Kreinin [44] reported a case of recovery from pneumococcic meningitis with the combined use of serum and sulfanilamide plus operation on the frontal sinus, and we have recently seen a similar instance in a child, but the case is not included in this series.

Pneumococcic meningitis was formerly almost as fatal as tuberculous meningitis. Gilmore and Sacks,[45] in review of the English literature on pneumococcic meningitis, cited Krentz and Witter as saying that infection of the mastoid due to the type III pneumococcus was twelve times as deadly as that due to Str. haemolyticus, which was 4 per cent fatal. Goldman and Herschberger,[46] by the use of an autogenous vaccine obtained from the mastoid at the time of operation, were able to reduce the mortality of infection due to the former organism also to 4 per cent.

In evaluating his experience in 15 cases, Hansen [47] concluded that Pneumococcus type III is more virulent in children than in adults. Harkness [47a] made the same observation. This conclusion is not borne out by our series, as shown in table 1, or by the experience of Maybaum and Druss [15] and Persky.[16]

Streptococcic Meningitis.—Streptococcic meningitis has responded well to drugs of the sulfanilamide group, and cures have been reported from the use of any one of them, though it now seems that sulfadiazine is the most effective. Convalescent serum may efficiently be used in combination with the drug. Rosenwasser,[48] in reporting cure through administration of sulfanilamide and operation in a case of otitic streptococcic meningitis, emphasized the importance of continuing treatment with the drug for some time after the disappearance of the organisms from the spinal fluid. In his case, when the fluid was sterile and the patient was recovering, the dose was greatly reduced; organisms quickly reappeared, with aggravation of the clinical picture. The dose was again increased, followed quickly by improvement, with ultimate recovery. Cunning and Guerry [49] claimed that sulfanilamide is still the

44. Kreinin, S.: Pneumococcic Meningitis with Recovery: Combined Use of Serum and Sulfanilamide, Arch. Otolaryng. **29**:371-377 (Feb.) 1939.

45. Gilmore, G. B., and Sacks, P.: Pneumococcic Meningitis: A Review of the English Literature, with a Report of Two Cases in Which the Disease Was Otitic in Origin, Arch. Otolaryng. **32**:1007-1022 (Dec.) 1940.

46. Goldman, J. L., and Herschberger, C.: Prophylactic Vaccination Against Intracranial Complications Following Pneumococcic Type III Mastoiditis, J. A. M. A. **109**:1254-1257 (Oct. 16) 1937.

47. Hansen, B. R.: Pneumococcic Types of Middle Ear Suppuration, Acta oto-laryng. **28**:340-351, 1940.

47a. Harkness, G. F.: Complications of Surgical Treatment of Acute Mastoiditis, Arch. Otolaryng. **32**:915-926 (Nov.) 1940.

48. Rosenwasser, H.: Otitic Streptococcus Meningitis with Recovery: Case Report, J. Mt. Sinai Hosp. **6**:203-205 (Dec.) 1939.

49. Cunning, D. S., and Guerry, DuP., III: Sulfanilamide and Its Derivatives, Arch. Otolaryng. **34**:809-819 (Oct.) 1941.

drug of choice when the virulent organism is the hemolytic streptococcus, but this is in disagreement with the observations of Spink. Of our 7 patients, 4 recovered and 3 died. The patients receiving sulfadiazine and sulfathiazole recovered (table 9). One patient received no treatment and diet. Four patients received sulfapyridine; 2 recovered and 2 died. Of the 4 patients who recovered, 3 had the benefit of surgical treatment.

INFLUENZAL MENINGITIS

With the use of chemotherapy and antiserum the mortality from this form of meningitis has been reduced. Sulfanilamide seems to have given the best results, although sulfapyridine and sulfathiazole have also been used successfully. Our 3 patients died.

It is essential to make an early diagnosis. This is sometimes difficult. Attention was first drawn to the pleomorphic character of influenza bacilli by Slawyk in 1899 and by many later writers.[50] Once the diagnosis is made, vigorous treatment should be instituted and continued until all danger of reappearance of the organism in the spinal fluid is past. Herein lies one of the chief advantages of chemotherapy, for administration of sulfanilamide, and particularly sulfathiazole, can be maintained after reactions have made serum treatment embarrassing and even dangerous. In connection with the early institution of vigorous therapy may be mentioned the tendency of this infection, untreated, to produce heavy exudate, with astonishing pressure about the base of the brain, thus accounting for the tendency to reappearance of bacilli after apparent initial success.

STAPHYLOCOCCIC MENINGITIS

This type is less frequently encountered than any of the meningitides previously mentioned. Fatality rates are likely to be higher. Of all the compounds of sulfanilamide, sulfathiazole has proved most efficient. Globulin-refined staphylococcus antitoxin may be given in cases of severe involvement.

In treatment of staphylococcic meningitis, Long [51] stated that sulfathiazole is inefficient because it does not readily pass into the spinal fluid, but later Banks [52] reported success with the drug and, citing a

50. Noone, E. L., and Kennedy, P. J.: Influenzal Meningitis: Report of Two Cases with Recovery; One Case Complicated by Paroxysmal Tachycardia, J. A. M. A. **115**:2060-2063 (Dec. 14) 1940.

51. Long, P. H.: Clinical Use of Sulfanilamide and Its Derivatives in Treatment and Prophylaxis of Certain Infections: Wesley M. Carpenter Lecture, Bull. New York Acad. Med. **16**:732-750 (Dec.) 1940.

52. Banks, H. S.: Sulfathiazole in Cerebrospinal Fever, Lancet **1**:104-107 (Jan. 25) 1941.

TABLE 10.—*Comparative Results of Surgical and Nonsurgical Treatment in Fifty-Eight Cases of Otogenic or Rhinogenic Meningitis*

Year	Otogenic								Rhinogenic							
	Cured				Died				Cured				Died			
	With Surgery		Without Surgery		With Surgery		Without Surgery		With Surgery		Without Surgery		With Surgery		Without Surgery	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
1938	0	0	1	11	1	11	7	78	0	0	2	33	1	17	3	50
1939	2	20	0	0	0	0	8	80	0	0	0	0	0	0	3	100
1940	4	31	1	7	5	38	3	24	1	11	2	22	3	34	3	34
1941	4	67	0	0	1	16.5	1	16.5	1	50	1	50	0	0	0	0
	10		2		7		19	(39)	2		5		4		9	(20)

	Number of Cases	Otogenic Cured No.	Otogenic Cured %	Otogenic Died No.	Otogenic Died %	Number of Cases	Rhinogenic Cured No.	Rhinogenic Cured %	Rhinogenic Died No.	Rhinogenic Died %	Total
Total operated on (40%)	17	10	60	7	40	6	2	33	4	66	23
Total not operated on (60%)	21	2	9.5	19	91.5	14	5	36	9	64	35

mortality of slightly over 2 per cent, concluded that while Long's observation is true, the drug is sufficiently concentrated in the spinal fluid to be efficacious.

COMMENT

A more detailed knowledge of the pathways of infection from the head to the meninges offers the hope of a logical approach to this problem.

With the advent of chemotherapy the outlook in cases of bacterial meningitis has improved in spectacular fashion. The results in our series, which show a 60 per cent rate of cure in cases of otogenic and a 33 per cent rate in cases of rhinogenic meningitis, are in general borne out by scattered reports throughout the country.

In those cases of bacterial meningitis in which no other focus is apparent the infection should be suspected of emanating from the ear, nose or throat, and all pertinent clinical and laboratory investigations should be made. Our series shows that 36.2 per cent of all meningitides are of otorhinogenic origin, but since autopsy occasionally has revealed unsuspected pathologic conditions in these areas, with no other demonstrable cause, we feel that the incidence is considerably higher. It is well to remember that sulfanilamide and its derivatives obscure not only the clinical features but the changes in the blood and the roentgenogram, so that it is best to perform the diagnostic measures before instituting chemotherapy. In any event, this factor must be properly evaluated.

We have seen 3 cases in which there was no apparent suppuration that could be determined by clinical or roentgenographic examination or by other laboratory means and the drum membrane looked perfectly normal, with only the vaguest history to suggest aural trouble, and yet on operation a completely broken-down mastoid was revealed.

Although cures have been reported from the use of chemotherapy alone, our best results have been obtained with the combination of surgical treatment, chemotherapy and administration of serum in properly selected cases. The drainage of a demonstrable or suspected focus is an accepted surgical principle. Brunner [8] stated that it is not safe to exaggerate the efficiency of drugs of the sulfanilamide group to the point of considering surgical intervention unnecessary. On the contrary, the advice given by Jansen many decades ago that the first symptom of meningitis is the last summons to operate is still an acceptable dictum. Operation should be carried out in every case of meningeal disease in which otologic or rhinologic involvement is suspected.

We have come to feel that intranasal operation on the sinuses in cases of meningitis is not as satisfactory as the external approach and

have now adopted the policy of performing the so-called radical ethmosphenofrontal sinus operation whenever these sinuses are involved.

Subsequent to experience in this series of cases, we have not left the mastoid wound open as heretofore but have filled the mastoid cavity with the sulfanilamide powder. We have not attempted to do this in our cases of sinus involvement.

The efficacy of drug therapy depends largely on the extent of the pneumatization of the mastoid, and this accounts for its relative inefficiency in the treatment of the well ventilated accessory sinuses of the nose, the air cavities permitting the collection of large amount of infected inert exudate.

Lindsay [53] described his experience in 4 cases of meningitis following infection of the middle ear and concluded that chemotherapy alone is most likely to succeed in cases in which meningitis has developed rapidly, by extension along vascular channels, before there has been gross destruction of bone, and in cases in which pneumatization is of the limited small cell type or there is partial sclerosis. In these types there is less probability of a focus of necrosis or abscess in the bone. Because a complication frequently develops suddenly, the protection afforded by chemotherapy should be provided as an initial step in the treatment. The masking of symptoms which occurs with chemotherapy apparently can be interpreted as a desirable effect, indicating a definite limitation of the infectious process. Lindsay made the point that the masking of symptoms by chemotherapy differs from the masking of pain by sedatives inasmuch as the disease itself is actually impeded.

With so many cases of recovery from meningitis, one must guard against presuming that the cure was effected by chemotherapy alone. One is likely to disregard the fact that a focus has been eradicated and surgical drainage established. Therefore, with the admitted benefit to be derived from chemotherapy, the patient is entitled to the additional advantage to be obtained from the elimination of a focus of infection.

It is to be emphasized that when there is bone necrosis, abscess formation or pooling of purulent material in air cavities, sulfanilamide and its derivatives will be ineffective unless the diseased areas are surgically evacuated.

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53. Lindsay, J. R.: Chemotherapy in the Treatment of Complications of Acute Middle Ear Suppuration, Ann. Otol., Rhin. & Laryng. **50**:159-177 (March) 1941.

CORRECTION OF FACIAL DEFECTS WITH LATEX PROSTHESES

TECHNIC

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A prosthesis is a substitute for a missing part of the body. Prostheses have been made of many materials, including celluloid, metal, vulcanite, gelatin and latex. Improvement in the construction of these devices keeps pace with the progress of plastic surgery. Thus patients today regain a passable appearance much sooner after they suffer their deformity than have those in the past. For deformities caused by war injuries and the loss of parts resulting from malignant disease prostheses have a special place. Time-consuming plastic operations may be deferred and planned while the patient wears the temporary restoration.

The indications for the use of such a cosmetic device are: (1) interim treatment, especially in cases of malignant disease; (2) inadvisability of surgical treatment because of coexistent disease; (3) likelihood of recurrence; (4) debility of the patient, and (5) probability that the cosmetic result of operation will be poor.

The ideal prosthesis should have certain characteristics:

1. It should be nonirritating to the skin or mucous membrane to which it is applied.
2. It should be flexible and move with surrounding parts.
3. It should be translucent enough to transmit color from within its own mass.
4. It must be easy to clean.
5. It must be ductile enough to be molded with a feather edge.
6. It should be light in weight.
7. It should be of such composition that it can be accurately colored, without the addition of too much cosmetic material later when it is worn.
8. It should be inexpensive.
9. It should not require elaborate or expensive apparatus to make.
10. The building technic should not be too complicated.

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The vulcanite, celluloid, metallic and papier-mâché devices required painting to blend them with the color of the skin. Warnekros,[1] Klocke [2] and Zinsser [3] described the use of gelatin base materials for prosthetic restorations. In this country, Lederer [4] described the method and made many facial restorations with such materials. Gelatin prostheses were a distinct improvement in cosmetic restorations because they possessed flexibility, lightness and translucency. The factor of flexibility permitted the attachment of the prosthesis to the face with adhesive materials, and the patient could wear the device with assurance that it was held firmly without attachment to a spectacle frame. It enabled the surgeon to dispense with provisions of undercut to provide hidden shelves for prosthesis attachment. Their translucency made these devices more lifelike than the opaque ones had been. However, they were not sufficiently resistant to warm temperatures, and on warm days they tended to melt.

Bulbulian [5] introduced the use of a prevulcanized latex compound for making prosthetic restorations. Peluse,[6] formerly of the department with which I am associated, demonstrated the detailed technic and practicability of the use of latex material for life masks. Liquid latex is so manufactered that the vulcanizing steps have been performed and the liquid may be poured into molds and requires only contact with air and warmth to complete the solidifying process. It may be thinned with a 28 per cent solution of ammonia or may be thickened with inert ingredients. Various kinds of liquid latex are available, lending the rubber to various industrial and artistic uses. I have found it indeed a useful prosthetic material, lending itself to many modifications and possessing properties giving it versatility as a prosthetic material. I have made restorations for the nose, ear, entire ocular orbit and finger, and, to my knowledge, am the first to have made prostheses of the breast and the calf of the leg.

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1. Warnekros: Deutsche Zahnärztl. Wchnschr. **21**:120 (March 14) 1918.

2. Klocke: Herstellung künstlicher Gesichtsprothesen, Med. Klin. **12**:566-568, 1916.

3. Zinsser: Ein einfacher Nasenersatz, München. med. Wchnschr. **60**:2734 (Dec. 9) 1913.

4. Lederer, F. L.: Prosthetic Aids in Reconstructive Surgery About the Head: Presentation of a New Method, Arch. Otolaryng. **8**:531-554 (Nov.) 1928.

5. Bulbulian, A. H.: An Improved Technique for Prosthetic Restoration of Facial Defects by Use of a Latex Compound, Proc. Staff Meet., Mayo Clin. **14**:433-439 (July 12) 1939.

6. Peluse, S.: A Simplified Technic for the Construction of Life Masks of Latex Rubber, Arch. Otolaryng. **31**:955-965 (June) 1940.

TECHNIC

Steps in Making a Prosthesis.—First, one must make a negative impression of the part containing the defect. Petrolatum is lightly applied to the part to prevent hair adhering to the plaster. The negative material may be ordinary molding plaster or an agar molding compound. Ordinary no. 1 molding plaster is mixed with a small quantity of potato starch. This is mixed with water in a rubber bowl and poured over the defect. If the defect is the nose or the ear, the cavity must be protected with cotton plugs to keep the negative mass from entering the body cavities. If used rather thin, the plaster gives

Fig. 1.—*A*, congenital absence of one external ear and external auditory canal. A rudimentary lobule was present. *B*, appearance with prosthesis. The rudimentary lobule should be removed or moved posteriorly to form a pedunculated button for better attachment of the prosthesis.

finer detail. The patient should be informed that the plaster warms as it hardens. Most patients find this a rather frightening experience unless they are constantly reassured.

When hardened, the plaster is removed and painted with a very thin coat of petrolatum or dental separating compound. It is then ready for the casting of the positive in hard plaster. Mixing potato starch with plaster gives the negative mold the property of disintegrating more easily when the negative and positive molds are separated. This is done by immersing the molds in hot water. The starch helps the breaking-up process of the negative by swelling. Another negative mass

which is useful, especially when a member such as the ear, with many undercuts, is cast, is the agar compound material. Such materials are available commercially and can be duplicated by compounding the following formula:

Powdered agar	4 ounces (124.4 Gm.)
Zinc oxide	1 ounce (31.1 Gm.)
Phenol	5 grains (0.325 Gm.)
Glycerin	1 ounce (31.1 Gm.)
Shredded cellulose [6a]	½ ounce (15.5 Gm.)
Shredded cotton	15 grains (0.975 Gm.)
Rubber cement	5 ounces (124.4 Gm.)
Water	100 ounces (3,110 Gm.)

This is the formula of Clarke.[7] We made a batch of this formula almost seven years ago, regrinding it after each casting, and though we have used commercial mixtures since, the original mass is still useful. If the mass becomes too loose and tends to run, it may be heated, the flame removed and an additional ounce or two of rubber cement added. The negative mold made of such an agar mass must be reenforced with a coating of plaster. For most types of procedures the negative mold made directly in plaster is satisfactory.

To make the positive, molding plaster or hard dental stone is made into a thin paste by mixing with water in a rubber bowl. When of the consistency of thick, smooth cream, it is poured over the negative mold. The negative may be tapped gently, or it may be placed against a dental laboratory vibrator to remove bubbles which tend to be present. The plaster is then allowed to dry. The positive mold is then removed from the negative impression by immersing both in hot water, tapping or separating them with a stout knife. The positive is then gently cleansed and is ready for the sculpture.

The new part may be modeled in several mediums. Dental pink wax, heated and modeled with warmed instruments, is readily workable and can be modeled with fidelity to the smallest detail. Penetration of the hot wax into the pores of the plaster should be avoided. With added experience in the use of sculptor's materials, we have made models with clay, fine porcelain clay and plasticine. Modeling with sculptor's clay requires that the model be kept damp during the entire process of modeling. We have tried the use of delicate porcelain clay but find it rather doughy and difficult to handle. Domestic plasticine is a clay kept workable with vegetable oil or glycerin. The Italian plasticine, made with olive oil, is excellent. The missing part is modeled in clay

6a. Shredded cellulose is obtained from the filling material in sanitary pads.

7. Clarke, C. D.: Molding and Casting, Baltimore, John D. Lucas Company, 1938.

and smoothed to a feather edge. If it is a nose, the nostrils must be formed with great attention to detail and hollowed out as deeply as possible, to allow sufficient airway. If the orbit and the eye are to be replaced, the glass eye, properly matched, should be available for insertion. More of the sclera of the glass eye should be discernible than is visible in the normal eye; otherwise the artificial lids will seem to be closed too much. Once the prosthesis is satisfactorily modeled in the clay or wax as to size, position and artistic fidelity, it should be very slightly enlarged in all of its dimensions to allow for some shrinkage

Fig. 2.—*A*, congenital unilateral absence of the ear. *B*, appearance with latex prosthesis. The restoration is as translucent to light as is the normal ear.

of the rubber later. It is then ready to have the pores of the skin reproduced.

To reproduce the pores and the fine tracing of the skin, a small amount of fresh mixed plaster is poured over the skin of the patient or of any volunteer, dabbing it over an area about 1 or 2 cm. in diameter. As it hardens, a wooden applicator is inserted into it for a handle. When hard, it is removed and can be used as a tampon or impressor to stamp gently the pores and delicate skin tracery into the model. This artifice does much to make the restoration a faithful reproduction, especially pleasing in making noses. It is important at this stage to obtain a definite feather edge on the model, taking advan-

tage of every natural wrinkle and fold of visible skin to blend the restoration into its surroundings.

In completing the mold, "keyholes," or small shallow depressions, are dug in two or three places near the edge of the positive mold, to insure an accurate fit of the parts of the mold later. It is very lightly coated with petrolatum, avoiding the sculptured parts. This positive plaster mold will later be the bottom portion of the two or three piece mold. The top half of the mold is now ready to be poured. Another batch of hard plaster is mixed, and it is poured carefully over the sculptured part and the bottom portion of the mold. The bottom portion is gently tapped to prevent air bubbles from remaining, and the plaster is allowed to harden thoroughly. The parts of the mold are then carefully pried apart and the clay washed out.

Construction of a passage for the poured rubber must be made. A A conical hole is drilled through the bottom half of the mold to allow the rubber to be poured. The passage varies from about ¼ inch (0.63 cm.) in diameter for a small prosthesis, such as an ear or a nose, to a ¾ inch (1.9 cm.) canal for a breast, calf of the leg or other large deficiency. It is dug into the bottom portion of the mold so that its approach is from the posterior, or hidden, side of the restoration. The mold is now thoroughly dried, because the air content of the plaster is depended on to "cure" the rubber.

The type of rubber to cast with is important. For delicate structures, such as the orbit of the eye, the ear or the nose, we use orthotex,[8] a brand of prevulcanized liquid latex which is thin but may be thickened by adding zinc oxide, after the method of Bulbulian. About 1.5 Gm., mixed into a thin paste with 3 cc. of distilled water, is added to each hundred cubic centimeters of orthotex. This is then suitably colored. For larger prostheses, we use rubberspra,[9] a thicker type of latex, which shrinks less and produces a somewhat more solid product. It may be thinned with a 28 per cent solution of ammonia.

Color is a vitally important adjunct in achieving good results. Application of the color to the surface of the prosthesis only adds to its artificial appearance. Blending the color into the liquid before pouring lends the restoration a color which is the nearest approach to that of the natural skin and does not impair the translucence of the prosthesis. As the skin takes its color from the flesh beneath it, so does the latex transmit the color from within its mass to the surface. In addition to the congo red and Biebrich III red dyes which are added to the rubber for color, we have searched for other tints to add to the liquid mass to make the restoration more skinlike in color. We were especially anxious to find browns and yellows which would not be

8. Manufactured by the Orthotex Company, Milwaukee.
9. The Stewart Art Products Co., Lake Geneva, Wis.

bleached out by the ammonia contained in the rubber. We have found that inert or ground colors may be added without too much bleaching action. We now add 3 to 5 drops of cadmium yellow and 2 to 5 drops of burnt umber to each 30 cc. of liquid latex before pouring. We feel that this makes the color of the part much more lifelike than the addition of the red dyes alone. Other tints we have found useful are bismarck brown in a 1 per cent solution and trinitrophenol in a 0.5 per cent solution. A few drops of each are sufficient to tint 30 cc. of latex a desirable shade. Some useful powder colors are manganese dioxide, chrome yellow and barium chromate yellow.

Fig. 3.—Modeling the ear in fine porcelain maker's clay on the plaster positive.

In pouring the mold, a strip of adhesive tape is placed over the lip of the jar, thus permitting the liquid to be poured only in a thin stream and keeping the bubbles from the top of the liquid rubber from entering the mold. Liquid latex should never be poured without first being strained. The visible surface of the prosthesis is now down, as the pouring is done from behind, into the posterior aspect of the prosthesis. Thus gentle tapping of the mold will send bubbles upward or to the back of the prosthesis. Gentle shaking by a dental laboratory vibrator is ideal. The mold is set aside from a quarter to half an hour, and then the excess rubber is poured out. A thin layer of rubber will have coagulated against the plaster of paris jacket. If this is thick enough, about ⅛ inch (0.32 cm.) thick, it is placed in an oven at about 150 F.

(65 C.) for twelve to twenty-four hours. If it is not thick enough, the liquid may be poured into the mold a second time, to add to its thickness.

After a day or two of drying, the mold is opened, and the prosthesis is inspected for bubbles, color and shrinkage. If the color is not quite right, an alteration of color is made, and a new pouring becomes necessary.

For a testing board, a plaster of paris slab, about 1 or 1½ inches (2.5 or 3.8 cm.) thick, is useful in pretesting the color of the liquid rubber mass before pouring. It eliminates much, but not all, of the necessity for repourings. When the liquid has been colored, a pre-

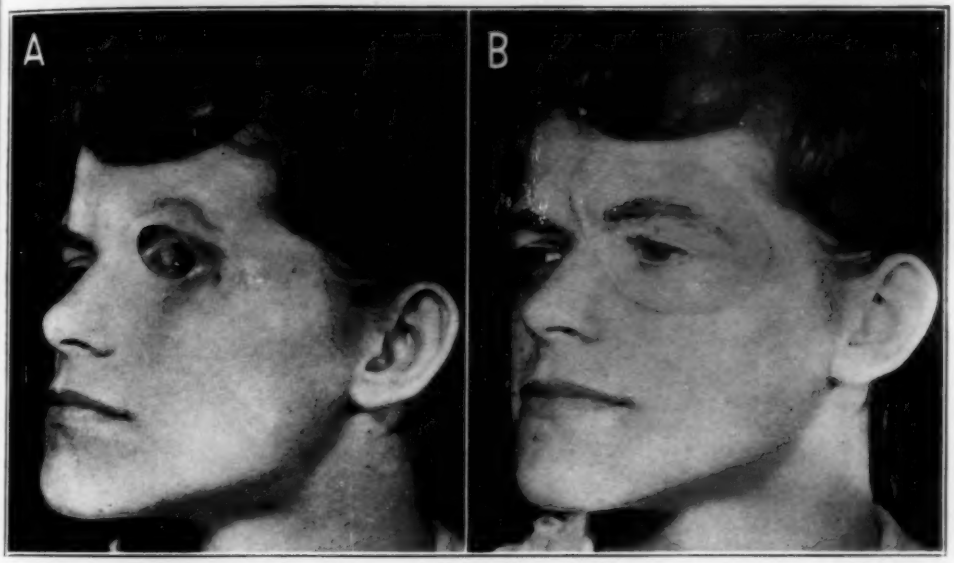

Fig. 4.—*A*, surgical exenteresis of the entire orbit. *B*, appearance with prosthesis.

liminary test for color is carried out as follows: A small quantity of the liquid is swabbed or painted on the dry, warm plaster slab. It dries in a few minutes, and then one can roll the thin sheet of rubber off the board with the finger and inspect the coagulated, rolled rubber for color. This is not the exact color of the finished rubber casting but, with a little experience, furnishes a better means of foretelling the color of the finished prosthesis than do the usual trial and error methods. Minor changes of color are then made before pouring the mold, until the sheets of rubber as they are rolled off the plaster board most nearly approach the color needed for the restoration. We have found the plaster board a time saver. Once the color is satisfactory, the liquid rubber is poured into the mold.

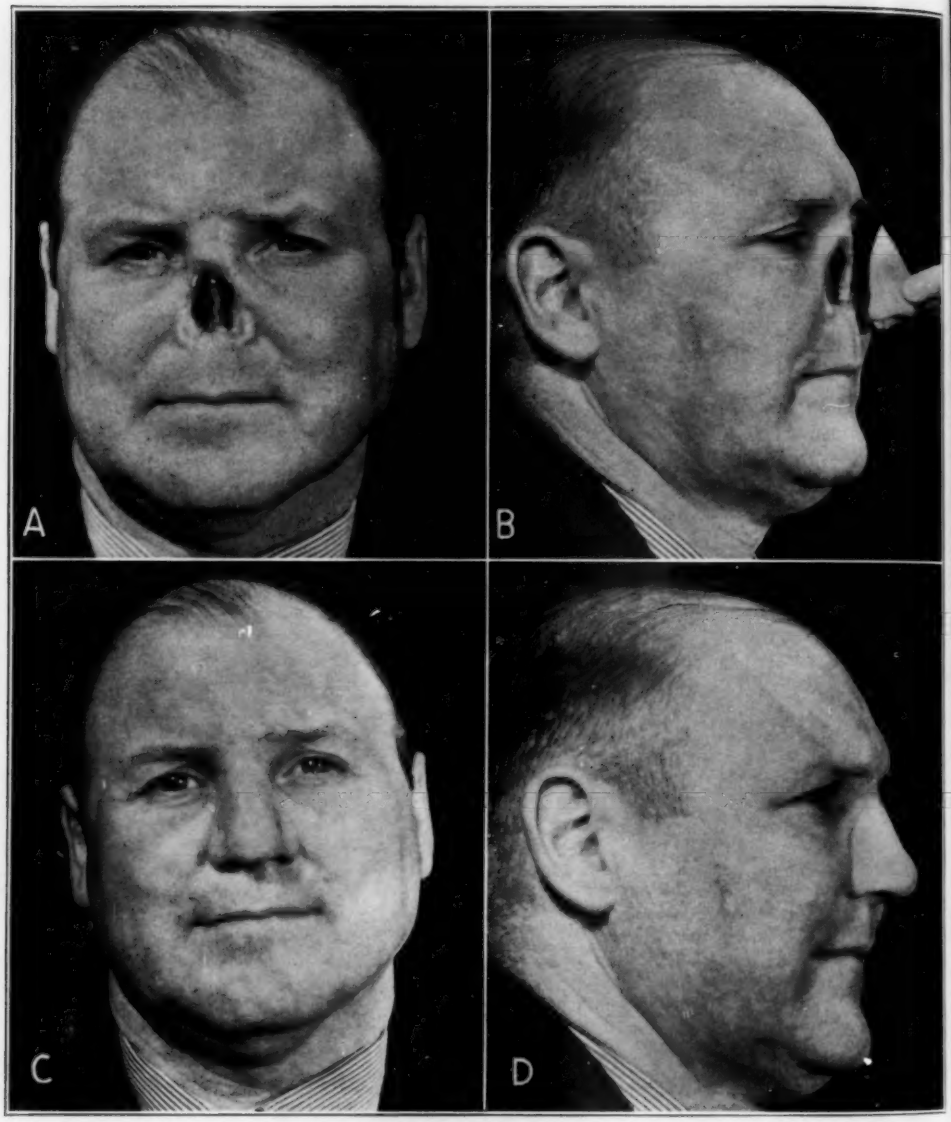

Fig. 5.—*A*, face after surgical ablation of nasal carcinoma. *B*, *C* and *D*, appearance with prosthesis.

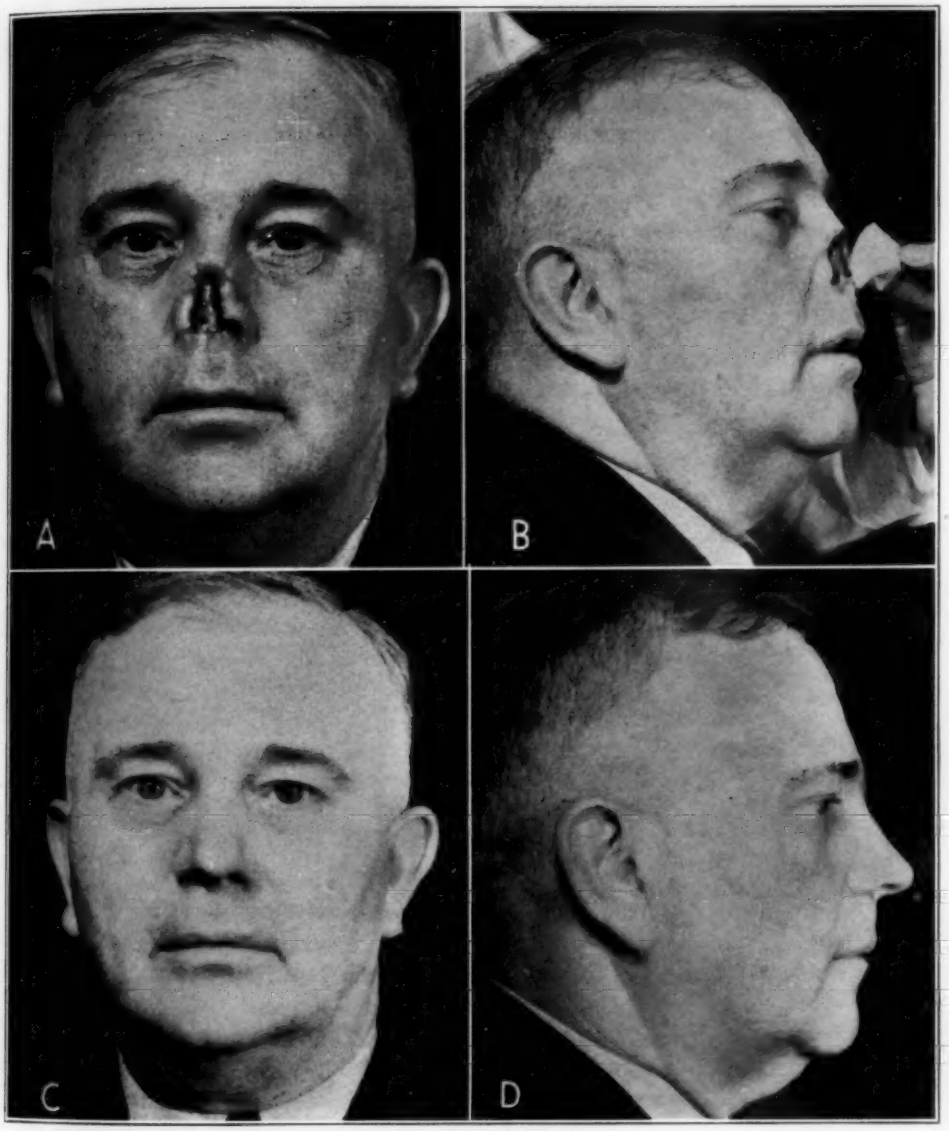

Fig. 6.—*A*, face after surgical removal of nasal carcinoma. *B*, *C* and *D*, appearance with prosthesis.

Curing the rubber is the next step. The rubber is self vulcanized. No additions are necessary to cause coagulation except to expose the rubber to air. Thus, plaster of paris permits air to reach the rubber through the pores of the plaster. If the mold is placed in an incubator or a small oven, or even on a steam radiator, at about 150 F., the vulcanization process is hastened. A stove top oven, made of sheet metal and heated with an electric bulb, is practical. After twelve to twenty-four hours, depending on the size of the piece, the mold may be opened and the prosthesis removed.

Trimming the Prosthesis.—The edges of the prosthesis may be trimmed with cuticle scissors. When extensive cutting is necessary, we find an electric burning pencil practical. This is the type of stylus used for wood burning and has changeable points of various shapes.

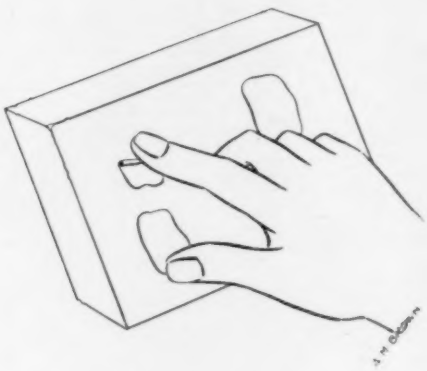

Fig. 7.—Rolling thin test sheets off the testing board to predetermine color of the restoration.

Seam lines may be obliterated with this heated knife edge, and irritating points on the skin side of the prosthesis may be trimmed away with it.

Attachment of the Prosthesis.—The prosthesis may be attached to a pair of spectacles or held with spring devices if skin recesses are available, or it may be attached with mucilage. An artificial ear may be fitted to the external auditory canal, if it is present. In 2 cases of congenital absence of the ear which we have encountered the canal is also absent, and the patients attach the ear with mucilage of good adhesive quality of the "prohesive" or "mastisol" type.

Mastisol adhesive is a compound containing styrax, gum mastic, benzene and ether. We have found it to be nonirritating and convenient to use. It is cleaned from the skin with benzene or ether and seems perspiration proof. It retains the prosthesis firmly, and prosthesis owners who use this adhesive material wear their restoration with assurance that it will remain well fixed. One patient is a foreman in

a stove works. He is able to keep his prosthesis clean with soap and water. Even gentle sneezing may be indulged in. A patient with an artificial nose soon masters the technic of drawing nasal secretion into the mouth to be expelled. Thus a minimum of "nose" wiping is necessary.

We have noticed that there seems to be less crusting within the nose while the patient wears his nasal prosthesis.[10] Van Alyea, in a previous article [11] and in a personal communication, expressed the opinion that amputation of the nose, whether pathologic or surgical, disturbs the normal physiologic activity of the nasal cilia. The patient's sense of well-being is enhanced when given the prosthesis. It is worth noting that some prosthesis wearers show such a wholesome attitude toward their restorations that they exhibit them proudly to relatives and intimate friends, much as some people display newly acquired dental bridgework. To observe how eagerly these patients accept their restorations is to be convinced that prostheses offer important psychic improvement to such persons.

This work was done under the guidance and stimulating advice of Dr. Francis L. Lederer, pioneer in the use of flexible translucent prostheses.

540 North Michigan Boulevard.

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10. We are about to make an interim prosthesis for a crusting mastoid defect and a crusting frontal sinus defect, and it will be interesting to see whether the crusting will be less when the protective rubber prostheses are worn.

11. Van Alyea, O. E.: The Acute Nasal Infection: Local Therapy Based on the Modern Conception of Nasal Physiology, Illinois M. J. **72**:336-341 (Oct.) 1937.

PROGNOSIS OF LARYNGEAL PARALYSIS FOLLOWING THYROIDECTOMY

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Injury to one or both recurrent laryngeal nerves during operation on the thyroid gland occurs frequently and usually results in paralysis of the vocal cord. When a patient who has sustained such an injury is examined in consultation, questions arise as to whether or not the voice will return to normal, when improvement should begin to become evident and what will be the future status of the laryngeal mechanism.

In an effort to answer some of these questions, histories of 504 patients who underwent partial thyroidectomy at the Johns Hopkins Hospital during the past five years were reviewed. In most, but not all, of the cases the larynx was examined by an otolaryngologist both before and after operation. In 32 patients the recurrent laryngeal nerves were injured at operation—a minimal incidence of 6.3 per cent. The histories of several other patients whose larynxes had not been examined, recorded a persistent hoarseness following thyroidectomy. These patients were not included in this study. Additional information about the voice and the appearance of the larynx was obtained by reexamination of as many of these 32 patients as could be induced to return to the clinic.

Thirty of the 32 patients with injury to the recurrent nerve had partial lobectomy on both sides; from each of the other 2 an adenoma was removed. Local anesthesia (procaine) was used for 5 of these patients; general anesthesia, for the others. Seventeen had hyperthyroidism; 14, nontoxic goiter, and 1, carcinoma. In 24 the paralysis was unilateral—on the right side in 14 and on the left in 10. In 11 of these 24 patients the nerve was injured on the side on which the lesser amount of thyroid gland was removed. In 8 both vocal cords were paralyzed, either partially or completely. In a patient whose right vocal cord was paralyzed at a previous operation, injury to the left recurrent laryngeal nerve at a second operation resulted in bilateral palsy of the abductor muscles.

Respiratory stridor during the operation was noted in only 1 patient, and an examination of the larynx made some days later showed paralysis of only one vocal cord. According to the operative notes, the recurrent laryngeal nerves were seen in only 2 of the 32 patients in whom the nerves were injured. In each case it was the left nerve which was

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exposed and identified and the postoperative examination showed paralysis of the right cord. In all of the 32 patients hoarseness of varying degree was noted immediately after the operation; in addition, 1 patient had marked stridor.

Seventeen of these 32 patients returned some months after operation for a laryngeal examination. After two or three months the speaking voice of patients with unilateral abductor paralysis was found to be definitely improved. After six months 13 of the 17 patients had fair conversational voice and could talk from room to room and over the telephone. One patient with unilateral paralysis had slight huskiness eighteen months after his operation. In 2 with complete paralysis on the right side and partial weakness of the abductor muscle on the left the hoarseness was still very marked a year later. In a patient with partial bilateral paralysis of the abductor muscle restoration of voice was due to complete recovery of the left recurrent nerve and normal movements of the left vocal cord. Fifteen patients were able to cough, another indication that the glottis could be closed tightly enough to allow intratracheal pressure to be increased. The 2 patients with hoarseness and bilateral weakness of the abductor muscle were unable to cough.

A lesion of the recurrent nerve causes a marked and usually permanent loss of volume of the speaking or singing voice. Ten of the patients in this series complained that after prolonged use, such as that required in dictating letters or in carrying on a long telephone conversation, the voice became progressively weaker and often faded to a whisper. This must indicate fatigue of the intrinsic muscles, which are overloaded in their effort to approximate the vocal cords. In 7 patients the speaking voice was decidedly lower in pitch after operation, and in 1 the pitch could not be controlled. The singing voice was lost in all but 4 and was weak and uncertain in these.

Dyspnea on exertion was not a prominent symptom but was present (possibly owing to other causes) in 2 patients with unilateral paralysis. In the 2 with bilateral weakness of the abductor muscle dyspnea was definite, and 1 of these patients had to have a tracheotomy later on account of increasing laryngeal obstruction.

Complete recovery of function of the vocal cord following trauma to the recurrent laryngeal nerve at operation is not to be expected. In 9 patients with complete unilateral paralysis there was no recovery of function of the abductor mechanism of the larynx. In 3 additional patients—1 with partial paralysis of the abductor muscle, a second with complete paralysis of the left and partial paralysis of the right vocal cord and a third with partial bilateral paralysis of the abductor muscle, the condition remained unchanged in subsequent examinations. A change from complete to partial paralysis occurred in 3 patients. Only

1 of the 17 patients who returned for later examination regained even approximately normal mobility of a completely paralyzed vocal cord.

Disappearance of hoarseness and the return of a satisfactory speaking voice are due to one of two mechanisms: a slight adductor motion may develop in the paralyzed cord, bringing this cord into approximation with the normal cord in the midline, or, in other cases, the normal cord may cross the midline and approximate the paralyzed cord sufficiently well to produce a fairly good speaking voice.

On the basis of this study one may conclude that the prognosis of laryngeal paralysis following injury to the recurrent laryngeal nerve during operation on the thyroid gland is that recovery of movement of the paralyzed vocal cord is unlikely but that recovery of a satisfactory conversational voice can be expected in about six months.

ATYPICAL FACIAL NEURALGIA

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SAN FRANCISCO

A large group of patients have atypical facial neuralgia characterized by severe boring, at times burning, pain, not limited to the distribution of a cranial nerve but occurring back of the eye, over the zygoma, cheek or nose and occasionally over the mastoid region and down the neck. It cannot be brought on by touching or moving the area and is not lancinating as is trigeminal neuralgia. This syndrome is often temporarily relieved by cocainizing the sphenopalatine ganglion, and Sluder, who first described the condition, thought that permanent relief was sometimes obtained by the injection of alcohol into that ganglion. I have seen patients relieved after cocainization but not after injection of alcohol.

Hypotheses as to the production of this pain are many. Davis and Pollock [1] stated the belief that they had disproved experimentally the existence of afferent sympathetic fibers. It was their opinion that stimulation of the efferent sympathetic fibers in turn stimulates the accepted pathways in the cranial nerves and produces the pain. Davis [2] expressed the belief that failure to relieve atypical neuralgia by section of the superior cervical ganglion and the sensory root of the fifth nerve shows that sensibility to pain on deep pressure is transmitted by the seventh nerve. Kuntz [3] stated the belief that there are no afferent fibers in the sympathetic nervous system.

Because in some cases of atypical neuralgia there is a distribution of pain which corresponds not to the area of any sensory nerve but rather to areas of arterial supply, Fay [4] has advanced the possibility of another mechanism. He stated that sectioning of various combinations of nerves had led to the impression that the vascular pain fibers of the head and neck find entry to the cranial nerves through the vagus, for the most

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Read at the Forty-Seventh Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Los Angeles, June 18, 1941.

1. Davis, L. E., and Pollock, L. J.: The Role of the Sympathetic System in the Production of Pain in the Head, Arch. Neurol. & Psychiat. **27**:282 (Feb.) 1932; The Role of the Autonomic Nervous System in the Production of Pain, J. A. M. A. **106**:350 (Feb. 1) 1936.

2. Davis, L. E.: Deep Sensibility of the Face, Arch. Neurol. & Psychiat. **9**:283 (March) 1923.

3. Kuntz, A.: The Autonomic Nervous System, ed. 2, Philadelphia, Lea & Febiger, 1934, p. 476.

4. Fay, T.: Atypical Facial Neuralgia, Ann. Otol., Rhin. & Laryng. **41**: 1030, 1932.

part near the bifurcation of the carotid artery. In addition, he suggested the probability that these fibers from the deep vascular tree of the face enter into the spinal cord in the upper thoracic and lower cervical regions. This was shown by inducing spinal anesthesia during an attack of the atypical facial neuralgia and observing the objective and subjective responses. The pain in the malar region was found to vanish as soon as anesthesia reached the first thoracic segment of the cord. Fay expressed the belief that it is futile to interrupt all the vascular pathways of pain as he had attempted to do by a large number of operations combining the sectioning of nerves and vascular pathways. He stated that when anisocoria to carotid stimulation is present and persists on the side of the neuralgia, an organic fixed basis above the carotid bifurcation is indicated for this symptom complex. This sign serves to differentiate the condition from the functional and distant reflex vasomotor disturbances associated with a similar distribution of pain.

Fay detailed three distinct mechanisms by which pain is transmitted from the structures of the face, head and neck: (1) superficial pain by way of the trigeminal and cervical nerves; (2) deep pain through the sensory branches and connections of the vagus nerve, and (3) tenderness and deep pain along the arterial tree, through the carotid sheath, to the cervical and thoracic portions of the spinal cord.

Reichert [5] also noted that relief could be obtained in what he defined as buccal neuralgia by interruption of the sympathetic fibers on the external maxillary artery. Thirteen of 17 patients for whom this method was used were relieved. Anatomically this may be correct, but the work of Woollard, Weddell and Harpman [6] showed that fibers similar to the sensory fibers in the superficial plexus of the skin, which give rise to pain stimuli, are found in the periarterial plexuses and in the adventitia of blood vessels.

By passing a fine needle through the skin over the brachial artery and obtaining the usual sensory response of a sticking sensation Waterston [7] showed that arterial walls have pain fibers. After two minutes, a period sufficient to permit the initial sensation to disappear, the point was passed farther, to come into contact with the wall of the artery. Immediate sharp pain was felt, localized and acute, the most unpleasant brought on from the test of any tissue, and on all occasions the response occurred immediately. The point was passed on and made to penetrate the arterial wall. This produced pain of a peculiar and distinctive character, with a sensation of nausea and faintness, but no pallor, sweating or actual sick-

5. Reichert, F. L.: Buccal Neuralgia: A Form of Atypical Facial Neuralgia of Sympathetic Origin, Arch. Surg. **41**:475 (Aug.) 1940.

6. Woollard, H. H.; Weddell, J., and Harpman, J. A.: Observations on the Neurohistological Basis of Cutaneous Pain, J. Anat. **74**:413, 1940.

7. Waterston, D.: On Pain, Lancet **1**:943, 1933.

ness was produced, and the pulse rate and volume of the radial pulse were unaltered.

Although these fibers may not be anatomically and embryologically associated with, or a part of, the sympathetic nervous system, it seems that the authors mentioned have shown that there are afferent fibers in the periarterial plexuses associated with sympathetic fibers which gain the central pathways via the cranial nerves or the dorsospinal routes.

There have been two other interesting contributions to the study of atypical facial neuralgia. The Mayo Clinic reported [8] the cure of 65 of 84 patients by desensitization with histamine. The workers were able to produce the typical headache by the injection of from 0.3 to 0.5 mg. of histamine phosphate. This headache was described as being accompanied at its onset by swelling of the temporal vessels, flushing of the side of the face, engorgement of the soft tissues of the eyes, profuse watering of the eyes and nose, conjunctival injection and plugging of the nose. This occurred on the side of the pain and was worse at night. An attack could be controlled by the intravenous injection of epinephrine hydrochloride and other vasoconstricting agents. It should be emphasized again that this type of neuralgia, i. e., the type described in the article from the Mayo Clinic, is identified by the engorgement and flushing of the side of the face and the fact that the neuralgia is worse at night. This is not the case with the neuralgias discussed previously.

Penfield [9] presented the concept that the dural nerves are a possible source of headache. He resected the anterior part of the falx cerebri and obtained a cure for a patient who had had supraorbital neurectomy, cervicosympathectomy on the left side, including removal of the stellate ganglion, and resection of the second, third and fourth cervical nerves. He reported the case of this patient, in which the lead to the diagnosis was an anomaly of the skull over the longitudinal sinus. This was seen in the roentgenogram.

The foregoing ideas have been proposed by clinicians on the basis of clinical experiences and sectioning of nerves. Fenton and Larsell,[10] as well as Blier,[11] have contributed much to the understanding of the mechanics of the sphenopalatine ganglion. They have expressed agree-

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8. Horton, B. T.; MacLean, A. R., and Craig, W. M.: A New Syndrome of Vascular Headache: Results of Treatment with Histamine; Preliminary Report, Proc. Staff Meet., Mayo Clin. **14**:257, 1939.

9. Penfield, W., and McNaughton, F.: Dural Headache and Innervation of the Dura Mater, Arch. Neurol. & Psychiat. **44**:43 (July) 1940.

10. Fenton, R.: Pathways of Referred Pain from the Nose, Am. J. Surg. **42**:194, 1938. Larsell, O., and Fenton, R. A.: Sympathetic Innervation of the Nose, Arch. Otolaryng. **24**:687 (Dec.) 1936.

11. Blier, Z.: Physiology of the Sphenopalatine Ganglion, Am. J. Physiol. **93**:398, 1930.

ment that vasodilators and secretory fibers reach the nasal mucosa from the vidian nerve through the sphenopalatine ganglion. These fibers seem to form a part of the great superficial petrosal nerve. The vasoconstrictor fibers appear to travel upward from the superior cervical sympathetic ganglion to and through the sphenopalatine ganglion. These data were established by sectioning of various nerves. The reticulospinal tract probably is the pathway whereby impulses from the sphenopalatine ganglion reach varying levels of the spinal cord. Allen [12] showed that this tract carries vascular reflexes. Christensen [13] also presented studies on the sectioning of nerves of the nasal autonomic system.

The case report presented here is that of a patient with atypical facial neuralgia, which was relieved by cocainization of the sphenopalatine region.

REPORT OF A CASE

A woman 41 years of age had pain, sharp and burning, back of the left eye, over the cheek and down the back of the left side of her neck. Whenever the pain was severe, extreme lacrimation of the left eye occurred. She had had simple hypertrophy of the thyroid gland. In 1928 the gland was removed surgically, and later a mild acidophilic adenoma of the pituitary gland developed, with signs of acromegaly. In 1937 roentgen therapy was administered for this. The neuralgia antedated the therapy and was not influenced by it. No coincidence between the onset of acromegaly and that of neuralgia was noted.

In September 1939 a submucous resection was done because of a very sharp septal spur which impinged on the left lower turbinate in its posterior portion. The relief of pain obtained for forty-eight hours was doubtless the result of the cocainization. Later, the injection of alcohol, via the posterior palatine canal, was tried, and relief was experienced for a few days.

Because Sewall [14] obtained a cure in a similar case by resection of the sphenopalatine ganglion, I decided to remove this ganglion through the maxillary sinus, which was done without difficulty, in August 1940. To facilitate the operation, the internal maxillary artery and several veins were ligated in the sphenomaxillary fissure. The infraorbital branch of the second division of the fifth nerve was laid bare and removed almost to the foramen rotundum but not to it. The region of the ganglion was removed en masse.

The following is the pathologist's report on the specimen removed:

"Gross Appearance: The specimen received is a piece of tissue from the sphenopalatine fossa, measuring 1 by 0.4 by 0.2 cm.; it is gray and soft.

"Microscopic Examination: The specimen consists of chips of bone and bone dust, ganglions, nerve fibers and arteries and arterioles. The section is stained with hematoxylin-eosin. Some of the ganglion nuclei are degenerated. The nerve fibers show much vacuolation. The arterial and arteriole walls are thick. In one area there are golden brown deposits of hemosiderin. There is a wide area of hyalinization surrounding some arterioles. These changes indicate chronic fibrosis."

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12. Allen, W. F.: Experimental Studies on the Visceral Bulbo-Spinal Pathway in the Cat and Guinea Pig, J. Comp. Neurol. **42**:393, 1927.

13. Christensen, K.: Innervation of the Mucous Membrane of the Nose, Tr. Am. Laryng., Rhin. & Otol. Soc. **42**:363, 1936.

14. Sewall, E. C.: Surgical Removal of the Sphenopalatine Ganglion, Ann. Otol., Rhin. & Laryng. **40**:79, 1937.

Immediately after the operation complete relief from pain was obtained for ten days and lacrimation was lessened. Anesthesia was present over the second branch of the fifth nerve. Pain was absent in the lower portion of the nose when a sharp test instrument was used. Strong pressure was felt as pressure.

Three months after operation (November) the severe pain was gone; rarely did the patient experience pain or pressure back of the eye, over the zygoma and down the neck and shoulder, but when this did occur, lacrimation accompanied it. There remained, however, an annoying burning sensation at the root of the nose and over the cheek, which areas were completely anesthetic to painful stimuli. Vasomotor response to epinephrine occurred. There was a continual feeling of "having a cold" in the affected side of the nose, but there were no visible signs of cold and the antrum was clean.

Feeling that the vasomotor control of the turbinate tissues and the location of the burning pain indicated that the vascular-borne afferent fibers along the external maxillary artery were at fault, I suggested the resection of a small portion of the artery, vein and immediately adjacent tissue. I planned first to inject procaine hydrochloride into this area during an attack. The patient felt so much better, however, that she preferred to wait, hoping for still further improvement.

In April 1941 the sense of deep pressure was present over the cheek and in the lower portion of the nose; sensory response to a sharp instrument was pressure, not pain. The former engorgement of the turbinates had subsided, and there was no visible difference between the two sides. Vasomotor response to epinephrine was normal, and the subjective sensation of nasal congestion was absent.

This last factor agrees with the findings of Vogel [15] (quoted by Sewall [14]), who observed that the vasomotor condition of the nose rapidly became normal after resection of the sphenopalatine ganglion. He was attempting to cure atrophic rhinitis by resection of the ganglion but failed to do so, as the vasodilation did not last, the function of the autonomic nervous system being taken over by the fibers via the external maxillary artery and probably by fibers of the internal maxillary artery which did not traverse the ganglion.

CONCLUSIONS

1. Resection of the sphenopalatine ganglion does not give complete relief in so-called atypical facial neuralgia of Sluder's or Reichert's type.

2. The periarterial plexuses carry afferent fibers probably not part of the sympathetic nervous system.

3. The return of vasomotor response after resection indicates that fibers efferent along the blood vessels take over this function. A section should be removed from the internal maxillary artery if the ganglion is resected. This would further cut down periarterial supply to the nasal tissues and the concomitant afferent fibers.

384 Post Street.

15. Vogel, K.: Histopathologische Befunde am Ganglion sphenopalatinum mit besonderer Berücksichtigung der atrophischen Rhinitiden, Ztschr. f. Hals-, Nasen- u. Ohrenh. **22**:507, 1929.

LOCAL USE OF SULFANILAMIDE AND ITS DERIVATIVES IN THE EAR, NOSE AND THROAT

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AND
BARNARD C. TROWBRIDGE, M.D.
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The favorable results from the internal use of sulfanilamide and its derivatives in suppurative conditions of the middle ear are widely known. However, little attention has been given to the possibilities of the local use of these substances in the treatment of infections of the ear, nose and throat.

As pointed out by Kolmer,[1] there can be no doubt about the effectiveness of local applications of sulfanilamide crystals in the prevention and treatment of infections of superficial wounds and burns and of local peritonitis. Kolmer stated further that it would appear the local administration of sulfanilamide and its derivatives for the prevention of infection is worthy of serious attention and wider application.

During the last two years we have observed many gratifying results following the local use of sulfanilamide and its derivatives in powdered form in suppurative and surgical conditions of the ear, nose and throat. Recently it has been demonstrated that a much higher concentration of sulfanilamide can be produced in the tissues of traumatic wounds by the local implantation of sulfanilamide powder than by any other method. Luo and P'an [2] made the striking observation that the concentration of sulfanilamide in the aqueous humor of the eye was considerably greater following local use of the powdered preparation than when large doses were administered orally or locally in liquid form. It is reasonable to assume that essentially similar high concentrations occur in the tissues of the middle ear and of the sinuses after local use of the powder.

The local use of sulfanilamide and sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) powder for purulent inflammation of the

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From the Otolaryngologic Services of the St. Joseph Hospital and Research Hospital.

1. Kolmer, J. A.: The Science of Chemotherapy, Arch. Otolaryng. **34**:1-6 (July) 1941.

2. Luo, T. H., and P'an, S. Y.: Concentration of Sulfanilamide in the Aqueous Humor of Human Eyes, with Special Reference to Local Application, Chinese M. J. **58**:167-176 (Aug.) 1940.

tympanum, particularly chronic otorrhea, has produced notable results in many cases. Acute and chronic suppurative otitis media, in which the perforation of the drum membrane allows the introduction of powder into the tympanum, is especially amenable to this type of therapy. The character of the otorrhea, the result of examination of the culture and other local physical observations determine which compound is the one of choice in the treatment of a bacterial infection. Sulfathiazole powder is preferable in otorrhea in which Staphylococcus and Pneumococcus are the predominating organisms. Powdered sulfanilamide is the compound of choice in otorrhea in which Streptococcus predominates. The local use of sulfanilamide and its derivatives in no way replaces the important procedures of keeping the ear clean and of removing the inciting factors.

Originally, before the powdered preparations of sulfanilamide and its derivatives were utilized, a solution of azosulfamide (disodium 4-sulfamidophenyl-2′-azo-7′-acetylamino-1′-hydroxynaphthalene-3′,6′-disulfonate) [3] was used in the ear and nose with good results. However, experience with the powdered preparations has shown them to be more effective than the liquid preparations. To date, clinical observations on the use of the soluble salt of sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) indicate that it also is less effective than the powdered preparations.

The technic of the insufflation of sulfanilamide or sulfathiazole powder in the ear is simple. Under good illumination the aural canal is thoroughly cleaned so that the membranous perforation is clearly visible. With a Lederman hard rubber insufflator, the powder is introduced into the middle ear. The powder does not cake or form hard masses and hence does not interfere with the drainage of the middle ear secretions. The insufflation is painless and in no way disagreeable to the patient. The frequency of insufflation depends on the amount of aural drainage. A profuse discharge may rapidly remove the powder from the tympanum. In this case daily insufflation is required at first.

Since the majority of otitic infections are due to Streptococcus, Staphylococcus and Pneumococcus, the use of the powdered forms of sulfanilamide and its derivatives in these conditions seems logical. As the powdered preparations have been used with much success in soft tissue wounds due to trauma, in osteomyelitis and postoperatively in surgical wounds,[4] it seems that their use in suppurative and surgical conditions of the ear would be of value. Although the powdered preparations of sulfanilamide and sulfathiazole are only slightly soluble in water, their use

3. This substance has been known as prontosil soluble, as prontosil and as neoprontosil.

4. Key, J. A.: The Use of Sulfanilamide and Sulfathiazole in Orthopedic Surgery, J. A. M. A. **117**:409-412 (Aug. 9) 1941.

locally has in no way produced undesirable reactions. Apparently the powder is soluble in tissue fluids and exerts its effect on the bacteria in this manner. Sulfanilamide is known to be a very diffusible substance, and its ability to penetrate the tissues of the body has been considered of importance in its efficacy against infection. Marshall and his associates [5] have demonstrated that sulfanilamide enters many of the tissues and fluids of the body. Because of the prolonged action of the powder, better results are obtained than when the liquid preparations are instilled into the ear. No toxic reactions have occurred from the use of the powders in the ears. The local use of sulfanilamide or sulfathiazole powder does not replace or interfere with the oral administration of sulfanilamide or its derivatives in suppurative conditions of the ear. Systemic medication with sulfanilamide and its derivatives should be carried on in conjunction with the local use of the drugs. Decrease in drainage and relief of symptoms have followed the use of the powder in chronic as well as acute suppurative infections of the ear. Sulfathiazole powder prepared in the form of an ointment is of distinct value as a local medicament for furunculosis of the external auditory canal. A 25 per cent mixture of sulfathiazole powder in an oxycholesterol-petrolatum base is applied locally and by tampon to the canal as often as is necessary.

The powder is especially useful in mastoidectomy wounds. It is liberally dusted into the wound before partial closure is made. Key and his associates [6] advocated the local implantation of sulfanilamide powder in contaminated wounds and in clinically clean wounds where infection is especially to be feared or would be particularly undesirable. Postoperative irrigation of mastoid wounds with an aqueous suspension of the powder minimizes the local surgical reactions. The local use of the powder in surgical wounds of the ear in no way affects the sulfanilamide level in the blood.

Cultures of material from the nasal mucosa and the sinuses in acute infections have shown the predominating organisms to be similar to those in the ear, namely, Streptococcus, Staphylococcus and Pneumococcus. In the majority of cases of chronic sinusitis the predominating organism is Staphylococcus. The compound of choice in the treatment of nasal infections depends on the etiologic factors involved. Acute nasal infections respond best to the local use of sulfanilamide powder. Chronic rhinitis and sinusitis respond best to the use of sulfathiazole powder. The technic of intranasal administration of the powder is easily and quickly accomplished. In acute and chronic rhinitis the nasal cavities

5. Marshall, E. K., Jr.; Emerson, K., Jr., and Cutting, W. C.: The Distribution of Sulfanilamide in the Organism, J. Pharmacol. & Exper. Therap. **61**:196-204 (Oct.) 1937.

6. Key, J. A.; Frankel, C. J., and Burford, T. H.. The Local Use of Sulfanilamide in Various Tissues, J. Bone & Joint Surg. **22**:952-958 (Oct.) 1940.

are first cleaned thoroughly. Powdered sulfanilamide or sulfathiazole is then insufflated into the nasal cavities with a Lederman powder blower. In order to avoid losing the powder from the nose, the patient is cautioned not to blow his nose for at least half an hour. The loss of powder by the passage of excretions into the throat occurs considerably more slowly than the loss of liquid preparations instilled into the nose. Marked relief follows the use of the powder, and a few applications will often do much to abort acute rhinitis. As a matter of fact, the application of the powder to the nasal mucous membranes when gently and skilfully done is generally less disagreeable to the patient than the application of a liquid by means of a swab, and because of the prolonged action of the powder, better results are produced. The powder does not remain as a foreign body. It is evidently soluble in the tissue fluids and does not cause local irritation by its presence. Sneezing rarely occurs after the insufflation of the powder.

Apparently, ciliary activity is not impaired by use of the powder. It was shown experimentally that the time of migration of the powder particles from Little's area to the orifice of the eustachian tube varied from ten to fifteen minutes. This migratory rate indicates little if any impairment of ciliary activity by the powder.

The powdered preparations have proved to be of value in nasal operations. Postoperative inflammatory reaction and bleeding are greatly reduced in submucous resection by the local application of sulfanilamide powder. The powder is insufflated with a straight-tipped powder blower between the mucosal flaps of the septum prior to suturing. If there is any value in the local use of sulfanilamide and its derivatives, it seems to be in such operations and in ethmoidectomy, in which the danger of meningitis is always present. Childrey [7] stated that there is definite indication for and real value in the local use of the sulfanilamide compound powders in facial plastic operations, in which asepsis is so important. This certainly applies in any constructive operation on the nose, such as submucous resection. The local use of sulfanilamide in ethmoidectomy not only minimizes postoperative complications but also hastens the healing and drying of the preexisting infection. Likewise, insufflation of the powder into the sphenoid and maxillary sinuses after operation is of value. Introduction of powder into the maxillary sinuses following antrotomy is simplified by using a curved laryngeal powder blower instead of the straight-tipped insufflator utilized for all of the other sinuses. Postoperative irrigation of the sphenoid and maxillary sinuses definitely aids the healing process. Irrigation of the sinuses with an aqueous suspension of sulfanilamide or sulfathiazole powder

7. Childrey, J. H.: Sulfathiazol Used with Cartilage Implants for Repair of Facial Defects, Ann. Otol., Rhin. & Laryng. **49**:709-712 (Sept.) 1940.

controls the surgical reactions and hastens healing. The irrigations should be continued at two to three day intervals as long as the washings reveal the presence of purulent secretions.

A rather simple but valuable procedure following local tonsillectomy is the application of the powder to the tonsillar fossae. The powder reduces capillary bleeding and lessens the postoperative reaction. It is applied with a powder blower immediately after tonsillectomy and at two day intervals up to eight days postoperatively.

With the development of the use of the powdered preparations it was necessary to determine what properties a powder should possess in order to be suitable for use in the ear and especially in the nose and sinuses. Powder introduced into the tympanum would have to be of such a character as not to cake or form hard masses. A powder which would harden and fail to disintegrate would act as a foreign body irritant and would obstruct drainage. These characteristics would offset any advantage arising from the local use of the powder. Likewise, in the nasal cavities it was necessary to use a powder which would not interfere with ciliary activity or cause undesirable reflex reactions. A powder which would act as a foreign body could not be used in the sinuses.

It was found that coarse powder was not adaptable for use in either the ears or the nasal cavities. Moderately fine or fine powder, of a 40 to 100 mesh character, proved to be the most satisfactory quality for use in these regions. Apparently, there was no advantage in the use of a sterile powder over that of a nonsterile powder. The efficiency of sulfanilamide and its derivatives is often impaired by sterilization. It is exceedingly difficult to sterilize these compounds without causing partial chemical breakdown. The inherent therapeutic action of sulfanilamide and sulfathiazole powder obviates the necessity for sterilization.

The success of sulfanilamide and its derivatives in combating infections due to Streptococcus, Staphylococcus and Pneumococcus has led to considerable research on the possible uses of the drugs. An increasing number of preparations of sulfanilamide and its derivatives have been developed, to determine their efficiency in various forms. Although little information has been accumulated concerning the local physiologic and therapeutic action of the powdered preparations, we have found the use of the drugs in this form to be of definite benefit in infections and operative procedures on the ear, nose and throat. Since the value of the powdered preparations in traumatic and operative wounds is well known, their use in infections and operations of the ear, nose and throat seems logical.

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NEUROEPITHELIOMA IN THE TEMPORAL BONE

INVASION OF THE PETROUS AND SQUAMOUS PORTIONS, WITH EXTENSION INTO THE MIDDLE AND POSTERIOR CRANIAL FOSSAE

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Embryonal types of tumors of neurectodermal origin, having as their most prominent histologic characteristic the formation of true rosettes, suggesting a relation to the primitive neuroepithelium, are seen most often in the retina and rarely in other parts of the nervous system. Outside the central nervous system certain tumors, described as undifferentiated sympathicoblastomas with true rosette formation, may resemble closely neuroepitheliomas in the neuraxis.

Neuroepitheliomas occurring along the course of peripheral nerves are rare. Stout,[1] in 1935, in a critical analysis of the literature, found only 3 cases which he considered to be instances of a tumor of this type. One was a case of tumor of the sciatic nerve reported in 1892 by Garré [2]; another, a case of a tumor of the median nerve associated with Recklinghausen's disease reported by Lanford and Cohn [3] and Cohn [4] in 1927 and 1928, respectively, and the third, Stout's own case of tumor of the ulnar nerve reported in 1918.[5] Stout [1] stated that the last 2 cases were also studied by Penfield. Geschickter,[6] in a study of the material at the Johns Hopkins Hospital, found 2 neuroepitheliomas in a series of

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1. Stout, A. P.: The Malignant Tumors of the Peripheral Nerves, Am. J. Cancer **25**:1, 1935.

2. Garré, C.: Ueber sekundär maligne Neurome, Beitr. z. klin. Chir. **9**:465, 1892.

3. Lanford, J. A., and Cohn, I.: Ependymal Neoplasm of the Median Nerve, South. M. J. **20**:273, 1927.

4. Cohn, I.: Epithelial Neoplasms of Peripheral and Cranial Nerves: Report of Three Cases; Review of the Literature, Arch. Surg. **17**:117 (July) 1928.

5. Stout, A. P.: A Tumor of the Ulnar Nerve, Proc. New York Path. Soc. **18**:2, 1918; M. & S. Rep. Presbyterian Hosp. **10**:236, 1918.

6. Geschickter, C. F.: Tumors of the Peripheral Nerves, Am. J. Cancer **25**:377, 1935.

900 tumors of the peripheral nerves. Andrus [7] reported a case of neuroepithelioma of the lung from Rossle's laboratory, which was removed by Sauerbruch, and Mendl [8] described a malignant "rosette tumor of the pleura." Bertrand, Blanchard and Vedel [9] found a neuroepithelioma along the course of the eighth intercostal nerve of an old horse.

A few instances of neuroepithelioma in the brain itself have been reported. Bucy and Muncie,[10] in 1929, reported a case of neuroepithelioma of the cerebellum and found in the literature only 5 acceptable cases of this tumor in the brain, including 1 reported by Bailey [11] in which the frontal lobe was involved. Naeslund [12] recorded the first case of neuroepithelioma in the cerebellum, and in 1938 Hirsch and Oldberg [13] reported the third. Cushing,[14] Tello and Herrera,[15] McLean and Lantiere [16] and Fresen [17] each added a case of cerebral neuroepithelioma. Elvidge, Penfield and Cone,[18] in a series of 210 cases of glioma, found 1 of neuroepithelioma in the posterior fossa, which traversed the skull and presented in the neck.

Rarely, a tumor having the structure of a neuroepithelioma arises in the nasal mucosa and possibly is derived from the olfactory placode.

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7. Andrus, W. D.: Ueber ein Neuroepitheliom der Lunge, Centralbl. f. allg. Path. u. path. Anat. **54**:195, 1932; Tumors of the Chest Derived from Elements of the Nervous System, J. Thoracic Surg. **6**:381, 1937.

8. Mendl, E.: Ein Fall von bösartiger Rosettengeschwulst des Brustfells, Frankfurt. Ztschr. f. Path. **50**:304, 1937.

9. Bertrand, I.; Blanchard, L., and Vedel, R.: Neuroepithéliome intercostal chez le cheval, Rev. neurol. **2**:783, 1935.

10. Bucy, P. C., and Muncie, W. S.: Neuroepithelioma of the Cerebellum, Am. J. Path. **5**:157, 1929.

11. Bailey, P.: Further Remarks Concerning Tumors of the Glioma Group, Bull. Johns Hopkins Hosp. **40**:354, 1927.

12. Naeslund, J.: Neuro-Epithelioma Gliomatosum, Upsala läkaref. förh. **31**: 193, 1926.

13. von Hirsch, E. F., and Oldberg, E.: Neuroepitheliom des Kleinhirns, Zentralbl. f. allg. Path. u. path. Anat. **69**:113, 1938.

14. Cushing, H.: Intracranial Tumors: Notes upon a Series of Two Thousand Verified Cases, with Surgical-Mortality Percentages Pertaining Thereto, Springfield, Ill., Charles C. Thomas, Publisher, 1932.

15. Tello, J. F., and Herrera, J. M.: Sobre un caso de neuroepitelioma cerebral, Arch. de neurobiol. **13**:661, 1933.

16. McLean, A. J., and Lantiere, S. R.: Cerebral Neuro-Epithelioma, Arch. Path. **21**:601 (May) 1936.

17. Fresen, O.: Ueber ein Neuroepithelioma cerebri, Beitr. z. path. Anat. u. z. allg. Path. **130**:157, 1939.

18. Elvidge, A.; Penfield, W., and Cone, W.: The Gliomas of the Central Nervous System, A. Research Nerv. & Ment. Dis., Proc. (1935) **16**:107, 1937.

Wohlwill,[19] in 1936, reviewed the literature covering the esthesioneuroepitheliomas (esthesioneuroblastomas) and described a case of his own.

Most of the tumors of the spinal cord resembling neuroepithelioma may be classified as types of ependymoma (Kernohan, Woltman and Adson[20] and Kernohan and Kernohan[21]). Some teratomas contain neuroepitheliomatous elements. Masson[22] discussed the possible origin from the neural crest of structures resembling neuroepithelium found in embryonal adenosarcomas of the kidney. He did not deny the possibility that the neural plate may be involved, as well as the neural crests.

The present case is reported with the permission of Dr. George L. Apfelbach.

HISTORY OF A CASE

History.—A white woman aged 49 was admitted to the Grant Hospital of Chicago on Nov. 13, 1937 in a semistuporous condition. She had received electrical treatments for trigeminal neuralgia at a sanatorium sixteen months before. In February 1937 she ceased having pain in the right side of the face. In March 1937 she noticed that the right side of her face was paralyzed. After this occurred, she began having difficulty in walking and complained of weakness and dizziness. A roentgen examination made at another hospital in October 1937 suggested the possibility of a cholesteatoma or meningioma overlying the petrous portion of the right temporal bone.

Ocular Examination.—Right Eye: There were proptosis and superficial ulceration of the cornea. The lids could not be closed. The pupil reacted to light and in accommodation. The red reflex was present. Tests of the external ocular muscles showed ophthalmoplegia involving the lateral rectus muscle. The ocular bulb did not move temporally beyond the midline. There was thought also to be some restriction of movement superiorly.

Left Eye: Iridectomy had previously been performed. The movements of the eye were normal.

Neurologic Changes.—The right side of the forehead could not be wrinkled, nor could a right nasolabial furrow be produced. There was diminished hearing on the right side. Pharyngeal reflexes were not present. The uvula was in the midline; when the patient said "ah" it deviated to the left. The right sternocleidomastoid and trapezius muscles had lost much of their volume. The right half of the tongue showed longitudinal furrows and deviated to the right when protruded. A definite Chaddock sign was present on the left side. The deep reflexes were accentuated and equal on the two sides. The impression from the

19. Wohlwill, F.: Contribuïções morfológicas para o problema dos tumores, Lisboa méd. **13**:489 (Aug.) 1936.

20. Kernohan, J. W.; Woltman, H. W., and Adson, A. W.: Intramedullary Tumors of the Spinal Cord: A Review of Fifty-One Cases, with an Attempt at Histologic Classification, Arch. Neurol. & Psychiat. **25**:679 (April) 1931.

21. Kernohan, J. W., and Kernohan, E. M.: Ependymomas: A Study of One Hundred and Nine Cases, A. Research Nerv. & Ment. Dis., Proc. (1935) **16**:182, 1937.

22. Masson, P.: The Role of the Neural Crests in the Embryonal Adenosarcomas of the Kidney, Am. J. Cancer **33**:1, 1938.

neurologic examination was that of paralyses of the fifth, sixth, seventh, eighth, ninth, tenth, eleventh and twelfth cranial nerves, due possibly to a meningoencephalitic process, a tumor or an abscess.

After admittance to the hospital the patient was able to make herself understood only with great difficulty. There were no palpable masses in the abdomen, the breasts or the neck. The thyroid gland was not enlarged. There was no lymphadenopathy and no evidence of any paralysis of the extremities. A large amount of yellow material began to discharge from the right nostril. There was a rapid rise in temperature, and the patient died four days after entering the hospital.

Autopsy Observations.—Base of the Skull and Brain: Protruding into the middle and posterior cranial fossae on the right side and arising from the ridge of the petrous portion of the right temporal bone was a lobulated tumor mass measuring 8 by 6 by 4 cm. (fig. 1). The surfaces made by sectioning the moder-

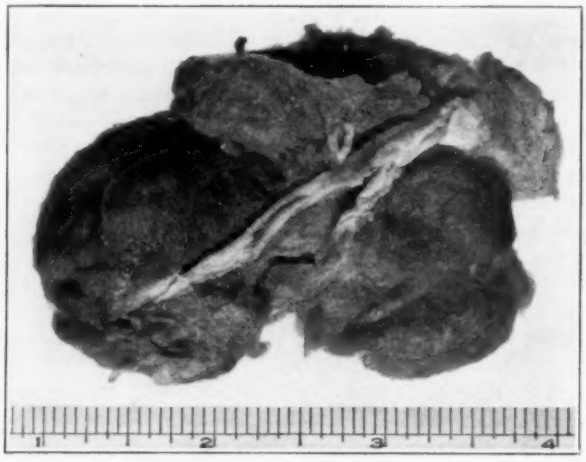

Fig. 1.—Tumor mass chiseled from the right temporal bone, showing the attachment of the tentorium cerebelli to the ridge of the petrous portion of the temporal bone.

ately firm, elevated areas were yellow-brown, mottled with yellow and purple-red areas measuring up to a pinhead in size. The tumor compressed the greater part of the right half of the inferior aspect of the pons. On the right side the brachium pontis, the flocculus and a portion of the cerebellar hemisphere were compressed so that the white matter was exposed (fig. 2). The fifth, sixth, seventh, eighth, ninth, tenth and eleventh cranial nerves were compressed at their entrance into the pons and medulla. The internal acoustic meatus was invaded by the tumor tissue, and the seventh and eighth cranial nerves could not be recognized in the mass. The dura mater over the posterior part of the petrous portion of the right temporal bone was absent. There were defects in the right half of the tentorium cerebelli through which the lobulated tumor tissue protruded. The tumor infiltrated the temporal bone, including the apex, the inner ear, the mastoid process, the tegmen tympani and the squamous portion, and invaded the greater wing of the sphenoid bone on the right side to a point within 1 cm. from the

foramen ovale. The infiltrations extended through the squamous portion of the right temporal bone, bulging into the subcutaneous tissue behind the helix of the right ear. The mass measured 20 by 10 by 5 mm. and was purple-gray on cut section. The jugular foramen and the sigmoid part of the transverse sinus on the right side were compressed by tumor tissue. There was no extension into the sella turcica or the sphenoid sinus. The ethmoid and maxillary sinuses contained no tumor tissue. There was no invasion of the right orbital cavity.

Microscopic Appearance of Tumor in the Temporal Bone.—There were irregular islands of tumor cells, which contained varying numbers of true rosettes. Some

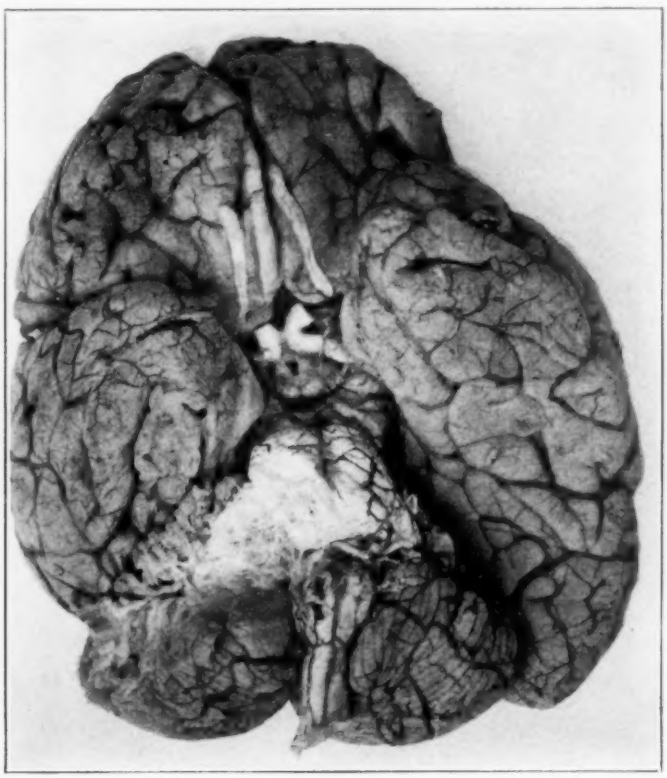

Fig. 2.—Photograph showing atrophy caused by the pressure of the tumor on the pons and the cerebellum.

of the cells lining the cavities were columnar or cuboidal and had round or oval nuclei with finely divided chromatin and a few clumps of chromatin, chiefly near the periphery. The cavities were also lined by smaller cells resembling lymphocytes, with very little cytoplasm. Among the cells, forming the rosette structures, were a few which had dark-staining, spindle-shaped or crescent nuclei (fig. 3). No well defined blepharoplast granules could be demonstrated with Mallory's phosphotungstic acid hematoxylin, Bailey's ethyl violet–orange G or Heidenhain's iron hematoxylin stain. Along the internal limiting membrane of a very occa-

sional rosette fine granules could be distinguished. These were interpreted as questionable blepharoplasts. As a control, the staining for blepharoplast granules was carried out with material from a malignant carcinoid tumor, in which well defined blepharoplasts could be demonstrated. No fine fibrillar material was found, only fine granular material, and this was retracted from the internal limiting membrane of the rosettes. This material did not take the mucicarmine stain. The Bielschowsky silver method did not show any stained tails or processes of the outer ends of the cells lining the cavities. No blood vessels were observed

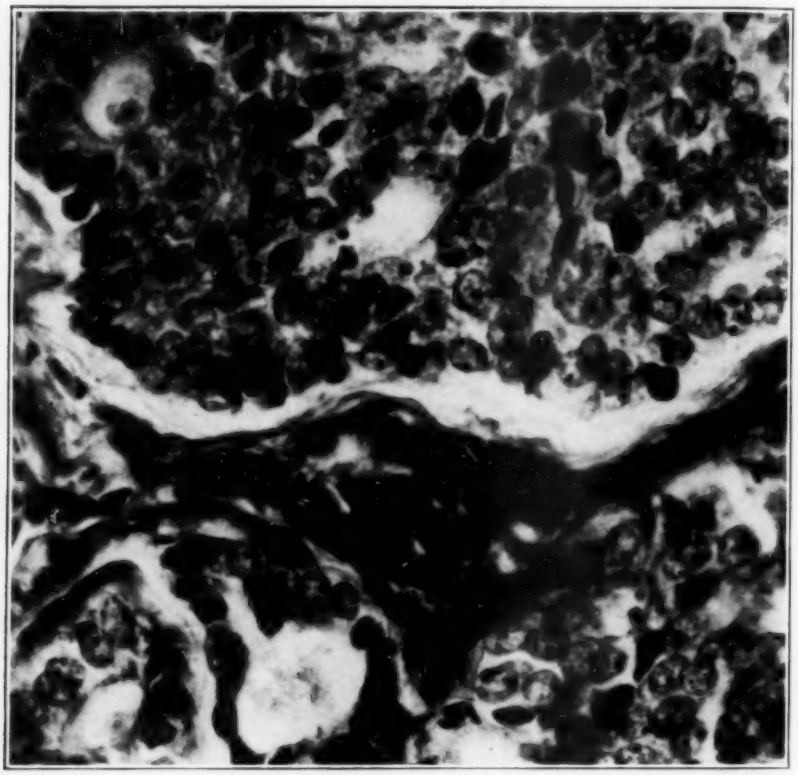

Fig. 3.—One of the smaller groups of cells, showing the true rosette formations and the light-staining and dark-staining nuclei. Iron hematoxylin stain; × 550.

within the cavities of the rosettes when studied with Mallory's phosphotungstic acid hematoxylin method.

In addition to rosettes with small canals, there were larger, irregular cavities which had a distinct internal but no appreciable external limiting membrane. Areas showing infiltrations of small groups of cells with true rosette formation were present. In many of these areas were larger, glandlike structures lined by cuboidal or lymphocyte-like cells. In a few of these structures blood vessels were seen. Some of the non-medullated nerve fibers showed invasion by tumor cells, with true rosette formation. Between the rosettes the cells were closely

packed together and had round or oval nuclei, which varied slightly in size. The smallest cells were about the size of a lymphocyte. Scattered throughout were elongated, spindle-shaped and crescent cells, the nuclei of which stained very dark with Heidenhain's iron hematoxylin stain. These dark cells stood out in marked contrast to the lighter, round or oval nuclei (fig. 4). Some islands of cells contained a few of these dark cells; others contained larger numbers. In some areas the dark cells were in preponderance and gave the appearance of a sarcomatous structure. Many of the islands showed large central areas of necrosis.

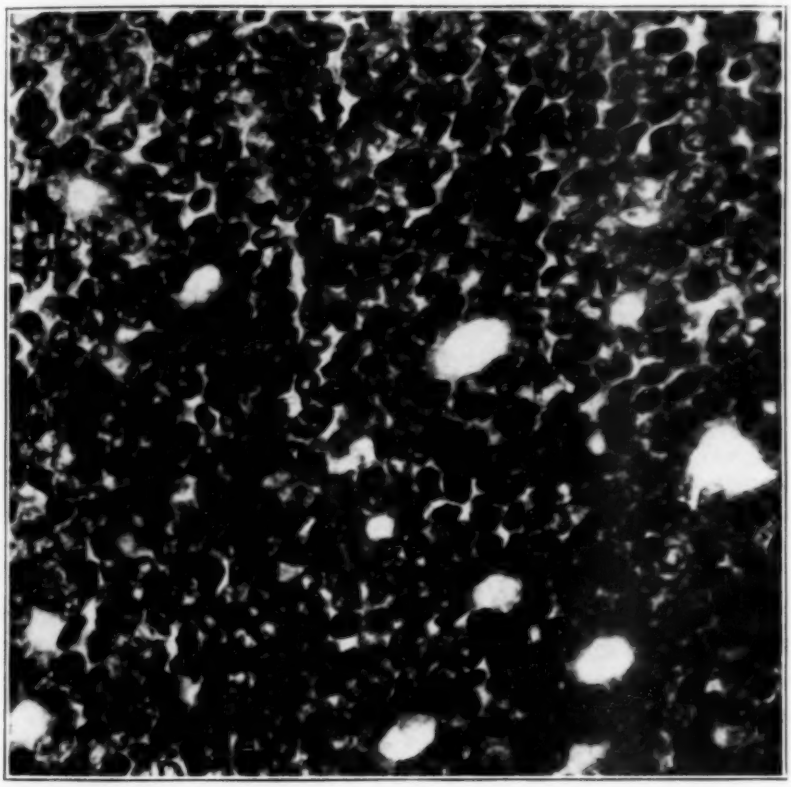

Fig. 4.—One of the large islands of tumor cells, showing the rosettes and the light-staining and dark-staining nuclei. Iron hematoxylin stain; × 350.

COMMENT

The diagnosis of neuroepithelioma can be established only by careful consideration of the criteria which have been generally accepted for this type of tumor. According to Bailey and Cushing [23] and Bielschowsky,[24]

23. Bailey, P., and Cushing, H.: Tumors of the Glioma Group, Philadelphia, J. B. Lippincott Company, 1926.

24. Bielschowsky, M., in Penfield, W.: Cytology and Cellular Pathology of the Nervous System, New York, Paul B. Hoeber, Inc., 1932, vol. 3, pp. 927 and 1085.

the neuroepithelioma is composed of cells resembling primitive spongioblasts, which are arranged around small canals and form true rosettes. Blepharoplasts may be present near the apex of the cells which line the cavities. The rosettes have an internal but no external limiting membrane. The cellular structure between the rosettes may resemble that of the cerebellar medulloblastoma. When no definite rosettes are formed, the cell bodies may be packed together to form a mosaic, with small irregular, empty spaces. The outer ends of the cells forming the rosettes may terminate in a tail or process, which can be stained only with difficulty by silver stains. The tissue in the case described by Lanford and Cohn [3] showed no cilia, blepharoplasts or processes, but Penfield classified it as a neuroepithelioma.

There has been a tendency in the literature to classify some ependymomas as neuroepitheliomas. Bailey [25] emphasized an important clinical distinction between the two tumors, which is based on the benign character of the ependymoma as compared with the malignancy of the neuroepithelioma. In the ependymoma and the ependymoblastoma the cells are arranged in a radiating fashion around blood vessels and form pseudorosettes. A small number of true rosettes may be formed in the ependymoma, but different degrees of differentiation are commonly observed in tumors of the nervous system.

Irregular, glandlike structures lined by columnar cells occurring in tumors with true rosette formation may suggest the rare medulloepithelioma. These structures are seen in malformations of the spinal cord and in teratomas. McLean and Lantiere,[16] in their report of a case of cerebral neuroepithelioma, stated that the irregular tubular and acinar formations without cilia or blepharoplasts might be thought to resemble a medulloepithelioma except for the fact that the glandlike structures did not possess an external limiting membrane. Davie [26] discussed the retinal and cerebral types of medulloepithelioma and stated that in the retinal type the irregular, glandlike elements do not show an external limiting membrane. Bailey and Cushing [23] made a separate group of the medulloepitheliomas, but in order to simplify the classification other investigators, such as Penfield [27] and Roussy and Oberling,[28] included them in the group of neuroepitheliomas.

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25. Bailey, P.: A Review of Modern Conceptions of the Structure and Classification of Tumors Derived from the Medullary Epithelium, J. belge de neurol. et de psychiat. **38**:759, 1938.

26. Davie, T. B.: Medullo-Epitheliomas of the Brain and Retina, J. Path. & Bact. **35**:359, 1932.

27. Penfield, W.: Classification of Gliomas and Neuroglia Cell Types, Arch. Neurol. & Psychiat. **26**:745 (Oct.) 1931.

28. Roussy, G., and Oberling, C.: Atlas du cancer, Paris, F. Alcan, 1931, pts. 9-10.

Occasionally there have been reported tumors of the sympathetic nervous system in which true rosettes were observed. The parent cell of the sympathetic nervous system is the sympathogonium. This cell is analogous to the carrot-shaped medulloblast of the central nervous system, described by Bailey.[25] The most primitive tumor of the general class of sympathicoblastomas has been called a sympathogonioma and is composed of small, dark-staining cells, suggesting an undifferentiated round cell sarcoma. Bielschowsky [24] mentioned that there may also be hollow spheres surrounded by sympathogonia. The more differentiated tumor is characterized by the formation of more or less fibrillar material, sometimes arranged in a central feltwork surrounded by cells and forming the typical pseudorosettes. Such a tumor is called a sympathicoblastoma. An analogous tumor in the cerebellum was termed a neuroblastoma by Wright,[29] and it was later classified by Bailey as a medulloblastoma. Scott, Oliver and Oliver,[30] in their review on sympathetic tumors of the adrenal medulla, reported a case with two types of rosettes, the true rosette and the pseudorosette. The cellular elements of the true rosettes were thought to be lemmoblasts, or primitive spongioblasts. However, many of the rosettes in their case contained the fine fibrillar material which is one of the characteristics of the partially differentiated sympathicoblastoma. No trace of such fibrillar material was observed in the tumor which we are reporting.

The histogenesis of the tumor involves elements derived from the neural tube, from the neural crest or from a placode. The ectodermal structures within the petrous portion of the temporal bone arise from all three sources. The efferent fibers of the facial nerve are derived from the basal plate of the neural tube, and the sensory fibers and the geniculate ganglion, from the neural crest. The epithelium of the inner ear and possibly part of the acoustic nerve are derived from placodes. As derivatives of the neural tube there are the neuroepitheliomas of the brain. The three tumors accepted by Stout as neuroepitheliomas of the peripheral nerves were said to be derived from the neural crest. Bailey and Herrmann [31] mentioned that such tumors may originate from Schwann cells, which are generally accepted as being derived from the neural crest. Tumors in the nose having the structure of a neuroepithelioma have been said to be derived from the olfactory placode (Wohlwill [19]), if such an analogy may be used. Masson [22] and Bielschowsky [24] suggested the possibility of a specialized neuroendodermal

29. Wright, J. H.: Neurocytoma or Neuroblastoma, a Kind of Tumor Not Generally Recognized, J. Exper. Med. **12**:556, 1910.

30. Scott, E.; Oliver, M. G., and Oliver, M. H.: Sympathetic Tumors of the Adrenal Medulla with Report of Four Cases, Am. J. Cancer **17**:396, 1933.

31. Bailey, P., and Herrmann, J.: The Role of the Cells of Schwann in the Formation of Tumors of the Peripheral Nerves, Am. J. Path. **14**:1, 1938.

placode in the intestinal mucosa to explain the ill understood nature of the argentaffine cells. Some of the argentaffinomas of the intestinal tract have true rosettes and blepharoplasts, which may bear some relation to the primitive neuroectoderm, although the recent studies of Popoff [32] linking the argentaffine cells with the life cycle of the mucous cells would not tend to bear out such an assumption.

SUMMARY

A malignant neuroectodermal tumor arising in and diffusely invading the temporal bone, with extension into the middle and posterior cranial fossae, has been classified as a neuroepithelioma.

32. Popoff, N. W.: Epithelial Functional Rejuvenation Observed in the Mucous Cells of the Gastrointestinal Tract and the Parietal Cells of the Stomach, Arch. Path. **27**:841 (May) 1939.

THE SPHENOID SINUS

A SAFE AND SIMPLE METHOD OF IRRIGATION

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It is usually a simple procedure to probe the sphenoid sinus through the natural ostium. In most cases it is only a matter of gentle manipulation and patience provided the parts are sufficiently anesthetized. However, at times it is very difficult and occasionally impossible. This is due to the fact that the natural ostium is variable in position and is not seen by anterior rhinoscopy except in rare circumstances.

The sphenoid sinus, in common with the maxillary, frontal and ethmoid cells, is an outgrowth of nasal mucous membrane. These evaginations of mucosa extend into and hollow out the bones from which they derive their names. The starting points, or narrow constrictions of the evaginations in the mucosa, persist in the adult as the ostiums, or openings, of the sinuses into the nasal fossae. When fully developed, the body of the sphenoid bone is hollowed out, forming a cavity. This cavity, or sinus, may be large or small, symmetric or asymmetric, regular or irregular, depending on the amount of spongy bone that has been absorbed. The size of the sphenoid sinus is important because if the sinus is small the walls are correspondingly thick, while in a very large sinus the walls may be only of eggshell thickness. This explains the danger of intracranial complications, particularly where the superior and external walls are concerned. These facts must also be borne in mind when probing the natural ostium or making an artificial opening in the anterior wall. The posterior wall is usually composed of thick, cancellous bone, which is fairly resistant. Occasionally, owing to enlargement of the sphenoid sinus and to the apparent pushing of the pituitary fossa into the sinus, the posterior wall is reduced to the thinness of paper. In such a case only very slight pressure with an instrument is necessary to perforate the posterior wall. Fortunately, this is a rare occurrence and is evident in a lateral roentgenogram.

The anterior wall of the sphenoid sinus slopes downward and backward and consists of two portions, the nasal and the ethmoid. The nasal

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The instruments and method of introduction were shown by means of a colored film.

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portion is about one third or more of the thin anterior wall close to the septum, which presents in the olfactory fissure and in the sphenoethmoid recess. The position of the natural ostium is not constant but is always situated in the nasal portion of the anterior wall, usually in the upper third and close to the septum. The depth of the sphenoethmoid recess seems to influence the situation of the ostium, as the deeper the recess the farther away from the septum is the opening. Occasionally, the ostiums on the two sides are not on the same level, and the distance from the floor of the sinus is also variable. However, in the vast majority of cases the opening is within 10 mm. of the roof of the nasal cavity. Only in exceptional cases can this be seen through the narrow slit available between the middle turbinate and the septum, so that it is necessary to depend on measurements and the sense of touch to find the aperture. Any enlargement of the middle turbinate or deflection of the septum increases the difficulty.

There are two methods of irrigating the sphenoid: (1) through the natural ostium and (2) by making an opening in the anterior wall of the sinus. Various instruments have been used for this purpose—some curved and blunt, others straight and sharp. Jacob, cited by Laurens,[1] and Sieur [2] recommended a no. 1 eustachian catheter to seek the natural ostium, while Watson-Williams [3] advocated a straight, blunt cannula to puncture the anterior wall, an instrument which in his hands proved very successful.

The method of puncturing the thin anterior wall has certain advantages over that of approach through the natural ostium. For instance, with a cannula inserted in place through an ostium which is very small and occluded by a swollen mucous membrane, it is impossible to apply suction, for, as Watson-Williams [3] said (page 175), there is no "bung hole." Making an opening into the sphenoid sinus is analogous to irrigating the maxillary antrum through the lateral nasal wall under the inferior turbinate. Once the parts are anesthetized, it requires only one or two minutes to insert the cannula into the sphenoid sinus, and material aspirated in this way is less liable to be contaminated.

In this connection, two instruments have been found helpful. The first is an indicator, or guide, which is placed in the nose at approximately the position of the natural ostium to ascertain proper distance and relation. The second is a cannula, which either is inserted through the natural ostium or is used to puncture the anterior wall close to the ostium. The same handle fits both instruments.

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1. Laurens, G.: Chirurgie oto-rhino-laryngologique, Paris, G. Steinheil, 1906, p. 578.

2. Sieur, cited by Watson-Williams,[3] p. 175.

3. Watson-Willliams, P.: Chronic Nasal Sinusitis, London, John Wright & Sons, Ltd., 1933, p. 176.

The indicator is a probe with a downward curve at the distal end and a handle that is easily removed once the probe is in place. The curved portion is serrated to hold absorbent firmly, and at the end is a small ball, which shows clearly in a roentgenogram. The length of the probe is 9.5 cm., and the straight end just protrudes from the nostril once the handle is disengaged. A raised marker 7 cm. from the ball indicates the distance from the anterior nasal spine to the anterior wall of the sphenoid sinus.

The sphenoid irrigator is a cannula with the same downward curve at the distal end as that on the probe. An elevated mark at 7 cm., as

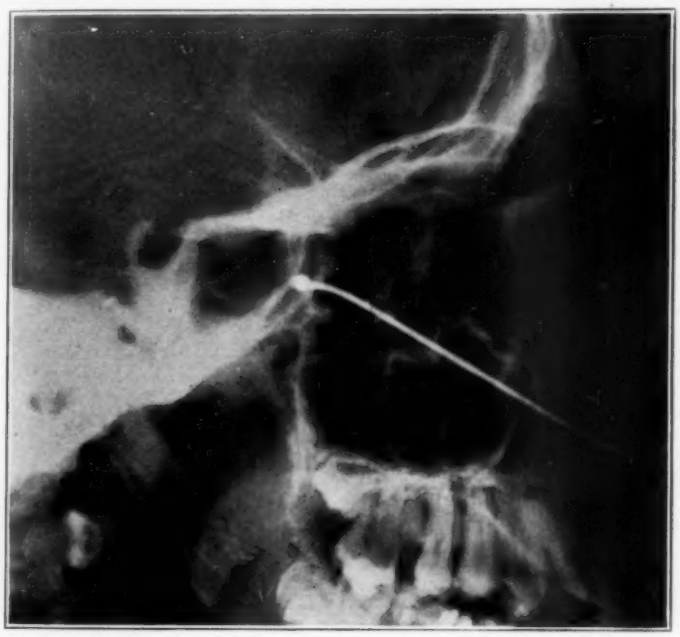

Fig. 1.—Roentgenogram showing insertion of the indicator.

has been mentioned, indicates the distance from the anterior nasal spine to the anterior wall of the sphenoid sinus in the average adult. With few exceptions, this distance, as noted by Zuckerkandl and later by Hajek,[4] is a fairly constant measurement.

The caliber of the lumen is sufficient to allow for the extraction of thick pus, which is impossible with the smaller, malleable cannulas. The curve at the distal end of the cannula is such that the tip will penetrate

4. Hajek, M.: Pathology and Treatment of the Inflammatory Diseases of the Nasal Accessory Sinuses, translated by J. D. Heitger and F. Hansel, St. Louis, C. V. Mosby Company, 1926, vol. 1, p. 83.

the thin anterior wall at the safest spot, which is usually within 10 to 12 mm. of the level of the cribriform plate.

The downward curve also insures entry into the lower half, or deeper part, of the sinus. (The difference between the upper, shallow portion of the sinus and the lower, deeper and much safer part has been stressed by Watson-Williams, page 179.)

A vertical tongue near the tip acts as a guard to prevent the instrument from penetrating too deeply into the sinus. This tends to avoid injuring the thin posterior wall which sometimes separates the sphenoid sinus from the pituitary fossa. Situated on the lower border of the cannula, this guard does not interfere in any way with vision.

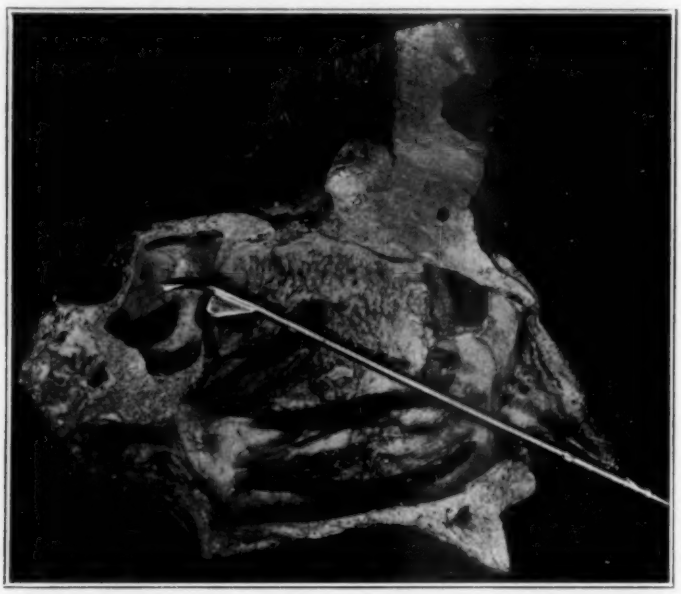

Fig. 2.—Sphenoid irrigator in place.

The proximal end for attaching to the syringe or suction is also turned downward slightly in order that an unobstructed view may be obtained along the barrel of the cannula. The handle is placed at a convenient angle so that when the tip of the instrument is in the sphenoid sinus the handle is perpendicular with the head erect or horizontal with the patient lying down.

After the nose has been thoroughly cocainized on the side to be examined, a small bit of absorbent cotton is wound tightly around the bulbous end of the indicator. This aids in holding the instrument in place. The probe is then adjusted in the handle and gently inserted between the middle turbinate and the septum up to the anterior wall of the sphenoid sinus.

To strike the anterior wall of the sphenoid sinus most textbooks suggest crossing the center of the lower edge of the middle turbinate diagonally. In using the aforementioned probe it is necessary to allow for the curve and cross the middle turbinate more anteriorly, that is, about 10 to 12 mm. below the top of the slit of the middle meatus. Once the instrument is in place the raised marker should be just inside the nostril. This shows the site of the anterior nasal spine. To release the handle, the screw on the side is turned slightly, leaving the probe fixed in position. A mental note is made indicating the spot where the instrument crosses the middle turbinate, or a diagram is drawn on the record and the patient sent to the roentgenologist for a lateral roentgenogram of the skull.

The position of the probe is immediately seen in the developed film. It is also noted whether or not the cannula, when inserted in place of the probe, should be elevated or lowered slightly to strike the region of the natural ostium. (It might also be mentioned here that the lateral view gives an approximate idea as to how much iodized poppyseed oil or other radiopaque solution is necessary to fill the sinus.)

When the landmarks and direction have been ascertained, the sphenoid cannula is used in place of the guide and an attempt made to find the ostium. If after a careful search it is impossible to probe the opening, very slight pressure is exerted in this region. This is usually sufficient to enter the sinus, as the area around the ostium is mostly membranous.

The importance of entering the sphenoid sinus as close to the septum as possible, in order to avoid injuring the lateral wall, with its adjacent cavernous sinus and optic nerve, has been stressed by various writers. However, the posterior part of the septum, which unites with the rostrum of the sphenoid bone, is thick, and it is necessary to turn the handle of the instrument slightly outward so that the tip is 3 or 4 mm. away from the septum before perforating the wall. This also tends to avoid the septal branch of the sphenopalatine artery, which sometimes is a vessel of considerable size. This artery crosses in front of the body of the sphenoid bone only a few millimeters below the ostium of the sinus.

At times it is perhaps more convenient to insert the cannula alone without the preliminary use of the probe. The instrument is placed in the nose between the middle turbinate and the septum, and an attempt is made to find the natural ostium. If this is impossible the instrument is held in the approximate position with a strip of adhesive tape over the bridge of the nose. The handle can either be removed or be left in place before the tape is applied. A lateral roentgenogram is then taken and the negative shows at once whether the tip of the cannula

should be raised or lowered in order to enter the sinus. It is most important to determine this before any pressure is exerted on the instrument.

Once the sinus is entered, this instrument is useful in outlining the cavity, in obtaining material for culture, in irrigation and treatment and as a guide in removing the anterior wall if this is found necessary.

These two instruments have been found of great value in cases of suspected sphenoiditis, both for diagnosis and for treatment.

Once the proper position of the probe is ascertained by means of a roentgenogram and the nose is anesthetized, it requires less than a minute to insert the cannula. After a little experience, it should be just as much a routine procedure to irrigate the sphenoid sinus when indicated as it is to puncture and wash out the antrum.

The instruments described in this paper are now manufactured by the Storz Instrument Co., 4919 Forest Park, St. Louis.

1390 Sherbrooke Street, West.

CALCULUS OF THE SALIVARY GLANDS

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Any abnormal lesion about the mouth, whether surgical or non-surgical, is of interest to the otolaryngologist, as such a lesion may be the cause of referred pain or other symptoms either of the ear or of the throat. Salivary calculi of all types, especially those of the parotid gland, are in the field of the otolaryngologist. The gratifying results of proper removal of such calculi merit serious consideration of this problem.

Case reports of salivary calculi are numerous in medical literature. The cause of these calculi, although still uncertain, has been variously ascribed to oral sepsis, dental caries, gout, systemic infections and endocrinopathies. Two main etiologic theories are to be considered. According to the first theory, the inflammatory process in the gland itself causes the formation of calculus; this assumption has received the support of such investigators as Kuttner, Hanzel and Kroiss. They expressed the belief that epithelium of the glands and their ducts, which is destroyed under the influence of salivary congestion (Graille), is likely to collect in lumps and form thus a nucleus for concrement formation. Additional depositis cause the stone to grow larger progressively. The inorganic salts present in the saliva can become crystallized out and laid down around the foreign material as a center.

According to the second theory, the calculus is of an inflammatory, particularly a bacterial, nature. This view is supported by the studies of Maas, Klebs, Galippe and, more recently, Naeslund [1] and Söderlund.[2] The last two investigators made extensive studies in this field, and each is cited by the other. In Söderlund's paper of 1920 he first associated formation of salivary calculus with actinomycotic infection and later reported 41 cases in which actinomycetes were present.

From the Department of Oral Surgery, University of Southern California School of Medicine.

Read at the Forty-Seventh Annual Meeting of the American Laryngological, Rhinological and Otological Society, Inc., Los Angeles, on June 17, 1941.

1. Naeslund, C.: Fortgesetzte Untersuchungen über die Speichelsteinbildung, Acta path. et microbiol. Scandinav. **6**:78-96, 1929.

2. Söderlund, G.: Die Speichelsteinkrankheit ("Sialolithiasis") und ihr Verhalten zu der primären und duktogenen Speicheldrüsenaktinomykose, Acta chir. Scandinav. (supp. 9) **63**:1, 1927.

In 1929 Naeslund reported his own experimental studies and his observations in a number of clinical cases. The entire study may be summarized as follows:

1. In 50 cases of salivary calculus all of the concretions examined histologically consisted of an actinomycete-like stroma.

2. In 11 out of 12 cases of salivary calculus examined bacteriologically pure actinomycete cultures were obtained from the stones.

3. Experimental formation of salivary calculus was achieved with actinomycete cultures which had been obtained from naturally formed salivary calculi.

4. Pure cultures were obtained from the saprophytic actinomyces, as well as from the ray fungus (Wolff-Israel), which is looked on as the causative organism of actinomycosis.

5. Pure cultures of the last-named actinomyces from salivary calculi induced typical progressive actinomycosis when injected subcutaneously in experimental animals.

If the actinomycotic theory of the formation of salivary calculus is true, one would expect to see at least an occasional case of actinomycosis of the gland itself. None of our patients showed any evidence of such a condition.

It is agreed that the submaxillary gland and its duct is the most frequent location of salivary calculus. It has been considered that the submaxillary gland is more susceptible to external trauma, bacterial infection and injury to the duct (Wharton's). According to the "Handbuch" of Henke and Lubarsch,[3] the high mucoid content of the submaxillary gland is responsible for the predominance of calculus in this gland. The saliva from the parotid gland contains no mucin. The authors assumed that the mucus content enhances the deposition of salts either on bacterial or on epithelial debris.

Certain anatomic peculiarities predispose the submaxillary gland to calculus formation. These include irregularities and diverticula in the excretory duct of the gland (F. Merkel). These irregularities serve as sites for secretory stasis and colonization of micro-organisms, with eventual stone formation. According to Zimmerman, the first of the diverticula is located 0.17 mm. below the caruncular surface; they are located rather close together, are numerous and do not begin to decrease in number until about 12 mm. from the orifice.

A survey was recently made of the cases of salivary calculus encountered during the past fifteen years in my clinics and in private practice.

3. Henke, F., in Henke, F., and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1929, vol. 5, pt. 2, pp. 25-36.

A total of 91 cases was seen; in 76 cases the calculus was in the submaxillary gland, in 11 in the parotid gland and in 4 in the sublingual gland. In 62 of the 91 cases surgical treatment was employed. In 52 cases calculi were removed from the submaxillary gland, in 6 from the parotid gland and in 4 from the sublingual gland. In 3 cases the submaxillary gland was excised.

There was a wide variation in age incidence, the oldest patient being 76 and 3 patients being 70 years of age. The youngest persons included in the series were between the ages of 11 and 13. The majority were in middle life. The ratio of males to females was 2 to 1.

Multiple calculi were encountered in 12 cases. The calculi varied from the size of a pinhead to 1¼ inches (3 cm.) in diameter.

The duration of symptoms was variable. In 1 patient intermittent symptoms were present for twenty-five years, and in 4 patients, for ten years; in 19 patients symptoms persisted for five years. Some had symptoms for only a few days. These short periods were especially evident when a calculus produced acute obstruction, manifested by painful swelling over the gland. In several cases of this type suppuration was a complication, as noted by purulent discharge from the affected duct.

The following interesting cases were encountered in this series: In 1 case a calculus 1¼ inches (3 cm.) in diameter was removed from the upper portion of the submaxillary gland through the mouth. Another case was one of bilateral submaxillary calculi, one of which was removed five years ago and one three years ago; the patient now has a bilateral recurrence. Of the 90 cases of unilateral calculus, 43 were on the left side and 47 were on the right side.

In 1 instance the removal of a submaxillary calculus was followed by mild obstructive symptoms of the parotid gland on the same side. These symptoms disappeared on the spontaneous discharge of several minute calculi from Stenson's duct.

Two cases were found in which the major portion of the submaxillary gland had been excised by other surgeons for relief of obstructive symptoms. In both of these cases I subsequently removed calculi by the oral route from the remaining portion of the gland. In 1 of these cases the gland had discharged through an oral sinus for nine months after external surgical intervention. In the second case, curiously enough, stones the size of wheat grains were found in the parotid gland five years later.

In a case of acute obstruction and suppuration of a submaxillary gland an external incision and drainage were carried out. After the acute process had subsided peroral removal of a calculus was attempted. The stone was exposed but was lost in the fascial planes of the floor

of the mouth and drifted inferiorly. The usual type of neck incision was then made and the stone located immediately above the hyoid bone. During the removal it crumbled into fragments and was removed only partially. Several days later the remaining fragments came out with the gauze drain.

Spontaneous discharge of the calculus occurred in 6 cases, either through the duct or through a fistula. In 1 case of parotid stone probing of Stenson's duct was followed by discharge of the stone.

Salivary calculus without infection may remain asymptomatic for years. Repeated attacks of soreness and swelling of the salivary glands, particularly when associated with eating or chewing, should always make one suspect the condition. Examination may show a red and edematous ductal papilla with a turbid or purulent discharge. The development of infection brings with it the usual signs and symptoms of acute inflammation or abscess formation. A calculus can frequently be palpated, or it may be located by needling, as suggested by Vilray Blair. I agree with New and Harper [4] that probing of the duct is of little help and may cause sufficient trauma to produce secondary infection. In 1 of my cases examination disclosed a painful, indurated Stenson duct with a diameter of 8 mm., the result of daily probings and irrigations over a period of weeks. As soon as treatment was discontinued recovery was prompt.

Proper roentgenograms usually reveal the stone. Injection of iodized poppyseed oil is of help, especially in case of the parotid gland. In the differential diagnosis one must remember that enlargement of a salivary gland may be due to benign and malignant tumors, cyst, actinomycosis, epidemic parotitis, postoperative parotitis, syphilis, tuberculosis, ranula, lymphadenitis and cellulitis.

Treatment necessarily varies with the stage of the condition. Since the main etiologic factor is recurrent obstruction, the duct should be kept open and the area free from infection. Oral hygiene must be maintained. If Wharton's duct is obstructed, anesthesia is best obtained by blocking the lingual nerve and making local infiltrations of procaine hydrochloride. In the floor of the mouth the tissues present a great range of motion, and fixation of the stone is difficult. Fixation is aided by upward pressure on the gland, the use of Havens' [5] hooks or a transfixion suture placed beneath the duct and beyond the stone.

In most cases it is possible to wait for the migration of a stone from the submaxillary gland into the duct. In the case of a physician patient this took one year, during which he had numerous obstructive attacks.

4. New, G. B., and Harper, F. R.: Chronic Inflammation of the Salivary Glands With or Without Calculi, Surg., Gynec. & Obst. **53**:456 (Oct.) 1931.

5. Havens, F. Z.: Removal of Calculi from the Submaxillary Ducts, S. Clin. North America **15**:1227 (Oct.) 1935.

In such a case it is best to operate when the calculus reaches the posterior border of the mylohyoid muscle because of the greater accessibility of this region. Further delay is of no avail. The duct makes a sharp curve at this point, impeding the further progress of the stone.

Careful dissection must be used to avoid cutting the lingual nerve or a deep-seated vessel. In general, smaller stones have been found to be more difficult to remove. The calculus is removed by an incision in the duct immediately over the stone, the incision being made in line with the duct. After removal, a small gauze drain is placed in the duct for a day or two, since the condition is often associated with mild pyogenic infection. Acute inflammatory symptoms must have palliative treatment before removal of the stone. Rest, liquid diet, hot mouth irrigations and cold external applications usually suffice, but occasionally chemotherapy is indicated.

The submaxillary gland should be removed if it is continually large and tender and contains the stone, if it is the seat of degenerative changes or if the stones are multiple. In approaching the submaxillary gland the incision should be made well below the border of the mandible; otherwise the branches of the seventh nerve supplying the depressor muscles of the angle of the mouth will be severed, producing a drooping lip.

Calculus of the parotid duct is usually removed by opening the duct from the mouth. If the stone is in the gland, the incision must be an external one, and great care must be taken not to injure the facial nerve.

CLINICAL APPLICATION OF VESTIBULAR TESTS

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INDIANAPOLIS

There are several reasons why vestibular tests have less clinical interest than auditory tests. First, the auditory tests are more pertinent and definitely more localized in the field in which the otologist works. Second, a vestibular test, when completely executed, takes considerable time. Third, the individual variation among patients makes standardization difficult. The highly nervous person, for instance, has a quicker reflex response than the slower, more lethargic person. Finally, at times contradictory findings are disheartening. Still these tests provide a source of much information which will be of value when otologists learn to interpret the findings.

Aside from the localization of various lesions in the end organ, the information obtained with these tests could be of great value to neurologists and neurosurgeons; it seems unlikely, however, that the data obtained by an otologist will ever contribute as much aid as that provided by the ophthalmologist, since the optic nerve runs a more unique intracranial course. The intimate relation with other vital structures, the greater susceptibility to the intracranial pressure and the desiccation of the chiasma all offer opportunities for study which the eighth nerve does not.

There have appeared in the literature many clinical reports which would lead one to believe that nearly any kind of intracranial condition can be diagnosed by a vestibular test alone. This is a little misleading, for it is difficult with our present capabilities to make a diagnosis of such a condition without using neurologic signs and symptoms. By the time a diagnosis can be made with neurologic methods, it is too late to be of much value, except to verify a previous diagnosis.

However, there are a few instances in which vestibular tests serve a useful purpose, particularly early in the course of a disease, before neurologic signs are well developed. In cases in which nystagmus, vertigo, instability, headache and deafness, either singly or combined, are present, differentiation by means of these tests between a peripheral and a central lesion is of great value. The table offers an outline of differential characteristics which is reliable in all except a few cases.

Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, Inc., Oct. 28, 1940.

The peripheral lesion may vary from transient toxic or pressure effects to complete destruction. That pressure in the middle ear is transmitted to the labyrinth is indicated by the experience of vertigo, as this is sometimes produced after inflation.

As a rule, when a part of the utricle and the canals are affected, the rest of the canals also are affected. This is true, particularly, in systemic conditions, such as infections, effects of drugs and blood dyscrasias.

From a vestibular standpoint, the end organ and the eighth nerve are to be considered as peripheral, and when a central lesion is referred to, extradural and intradural lesions are included. From early lesions which lie extradural but in close proximity to the dura, both branches

Differential Characteristics of Peripheral and Central Lesions

Peripheral	Central
Explosive onset	Slow onset
	Disturbed vision, headache
Dizziness, nausea, vomiting diarrhea	Dizziness, nausea, vomiting, diarrhea
Patient unable to arise	Patient up and around
Usually accompanied by ringing in the ears	Seldom accompanied by ringing in the ears
Spontaneous vertical nystagmus never present	Spontaneous vertical nystagmus may be present
Tests will not show responses in wrong direction	Patient may show nystagmus in wrong direction if wires are crossed
Romberg falling follows affected ear; vertigo does the same	Romberg falling follows same direction regardless of position of the head
Usually both the cochlea and the canals are affected	Nonresponsive canals may be present with good hearing
Horizontal canals usually the last to go out	Vertical canals may be out; horizontal canals normal
Usually there is harmony between nystagmus and vertigo	Nystagmus or vertigo may be changed
Compensation after 14 days	Symptoms persist; if anything, they become worse

of the eighth nerve are involved, and typical peripheral symptoms are produced. Tumors in the cerebellopontile angle are of this nature, and after sufficient enlargement takes place, pressure on the pons shows up in other locations, including the opposite eighth nerve.

The case hereinafter reported represents an advanced stage of an eighth nerve tumor with few neurologic symptoms and atypical vestibular symptoms. At some time during the progress of this case, the typical syndrome of cerebellopontile angle tumor might have been found, but by the time the patient reached the hospital, fifteen months had passed since the symptoms first appeared.

REPORT OF A CASE

CASE 1.—A white woman (C. S.), aged 18 years, was admitted Jan. 12, 1940 with the complaint of blindness in the left eye. Her illness began in October 1938 with prominence of the left eye; this slowly increased until she lost the sight in that eye.

Examination revealed a thin, poorly nourished but ambulatory woman who did not appear ill. She had no sight in the left eye with 4 to 5 D. choked disk and secondary atrophy (chalky white). There was also 4 to 5 D. choked disk in the right eye. There were vertical nystagmus on looking up, horizontal nystagmus on looking to either side, deafness in the right ear, absence of the pharyngeal reflex and cracked pot sound on percussion of the skull. There were unsteadiness on finger to nose test, a little awkwardness on rapid alternation of hands, a little unsteadiness of gait, questionable loss of the left ankle jerk and loss of the left achilles tendon reflex. The laboratory tests were all negative including serology except that roentgen examination after ventricular injection showed the left side to be slightly more dilated than the right and the third and fourth ventricles to be displaced to the left.

Vestibular tests showed marked loss of hearing in the right ear (90 decibels or more) and slight loss in the left (40 decibels) with no tinnitus. Spontaneously, the Romberg falling was to the left; there was no falling with the face turned to the right, and there was a tendency to fall to the right with the face turned to the left. There was no nystagmus on looking front or down; vertical nystagmus (mixed rotary and oblique to the left) was present on looking up, and horizontal nystagmus was present on looking to the right and left. When the patient was examined for past pointing, both arms touched.

The rotation test gave no response, neither nystagmus nor vertigo. After the right ear was douched for four minutes, no responses were obtained with the head erect or back. Both arms touched, and there was only slight vertigo. After the left ear was douched for four minutes, there was a slight suggestion of a horizontal nystagmus to the right with the head erect but not even a suspicion with the head back. Both arms past pointed an inch to the left with only slight vertigo. No galvanic test was done.

The diagnosis of tumor in the right cerebellopontile angle was verified at operation, when a large encapsulated mass was removed, despite exophthalmos and blindness in the left eye.

COMMENT

In these tests, the audiogram was made on a 6 A Western Electric audiometer in a semisoundproof room without a masker. The turning test—which should not be abandoned since irritation may result from it at times when caloric stimulation does not elicit a reaction—was performed at the rate of ten turns in twenty seconds. For the caloric test, flowing water (68 F.) through a Fowler glass irrigating cup with a small rubber extension to the center core was used. When galvanic stimulation is employed, a steadily increasing current is used, rather than a make and break current, with one electrode in the tragus and the other held in the hand on the same side.

It has been my experience that the nystagmus resulting from galvanic tests is rotary in normal persons, regardless of the position of the head, while Northington [1] stated that it is horizontal regardless of the position

1. Northington, P. O., in Fowler, E. P., Jr.: Medicine of the Ear, New York, Thomas Nelson & Sons, 1939, p. 427.

of the head and Best and Taylor [2] stated that it is mixed rotary and horizontal.

In considering intradural lesions, it has been customary to separate them from a vestibular standpoint into supratentorial and infratentorial. The structures above the tentorium are the cerebrum and its tracts and appendages (including the medial and lateral geniculate bodies), while below the tentorium are the medulla, the pons and the cerebellum. The mesencephalon passes through the tentorium and is made up of the cerebral peduncles and the corpora quadrigemina.[3]

It has been customary also to consider that hyperirritability on vestibular excitation is associated with a supratentorial lesion while hypoirritability occurs with infratentorial lesions. However, the reports from the current literature hereinafter noted show how contradictory the evidence from examination really becomes.

Güttich [4] reported 20 cases of cerebellar tumors, in 13 of which there was present a hyperirritable vestibular apparatus.

Brunner,[5] in analyzing 80 cases of tumor of the cerebrum and the cerebellum, observed normal responses in 30 per cent, hyperirritability with infratentorial lesions in 56 per cent and hyperirritability with supratentorial lesions in 67 per cent. The difference was not as great as might have been expected, but Brunner explained that the hyperirritability with infratentorial lesions was probably caused by irritation of the nystagmus centers by pressure on the brain stem. He stated also that the hyperirritability commonly encountered in supratentorial lesions is not all vestibular but is largely caused by irritation of the visual centers and tracts by pressure. He did not find complete lack of response in any of these cases.

Spiegel and Alexander,[6] in corroborating their previous experimental work on animals (in which they concluded there must be a bilateral cortical vestibular center in the temporal lobe) observed 192 patients. After eliminating effects of pressure and the cerebellum, they found that tumors close to the sylvian fissure caused vertigo more easily than those farther away.

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2. Best, C. H., and Taylor, N. B.: Physiological Basis of Medical Practice, ed. 2, Baltimore, Williams & Wilkins Company, 1939, p. 1370.

3. Ranson, S. W.: The Anatomy of the Nervous System, ed. 4, Philadelphia, W. B. Saunders Company, 1934, p. 34.

4. Güttich, A.: Otologic Experiences in the Observation of Brain Tumors, Ztschr. f. Hals-, Nasen- u. Ohrenh. **36**:78, 1934; abstracted, Arch. Otolaryng. **21**: 226 (Feb.) 1935.

5. Brunner, H.: Caloric Tests of the Labyrinth in Cases of Tumor of the Brain, Monatschr. f. Ohrenh. **70**:206 (Feb.) 1936; abstracted, Arch. Otolaryng. **25**:698 (June) 1937.

6. Spiegel, E. A., and Alexander, A.: Vertigo in Brain Tumors, with Special Reference to the Results of Labyrinth Examination, Ann. Otol., Rhin. & Laryng. **45**:979 (Dec.) 1936.

Barbier and Mayoux [7] reported 1 case of tumor of the right cerebellar hemisphere in which there was disturbed gait as the only neurologic symptom. The vestibular findings were normal excitability of both horizontal canals and inexcitability of the vertical canals of both sides. They regarded this as a syndrome characteristic of tumor of the cerebellum near the vermis.

Brunner [8] reported 3 cases of cerebral tumor which confirmed the findings of others that with frontal and frontotemporal tumors there occurs conjugate deviation of the eyes toward the slow component following caloric excitation instead of nystagmus to the opposite direction.

Leidler [9] implied that all vertigo is vestibular and probably a consequence of nystagmus. However, he expressed the opinion that the otologist has overstepped the mark in regard to equilibration, since many more reflex senses enter the maintenance of equilibrium than have been considered. He expressed the thought also that the old stumbling block of normal excitability of horizontal canals on rotation but complete inexcitability on caloric test is a sign of lesion of the central nervous system. Zacks [10] expressed the belief that this set of responses occurs with head injuries.

Güttich [4] confirmed the findings of Barré [11] and others that hyperexcitability prevails in cases of multiple sclerosis.

The vestibular excitability following injury to the head also has been under consideration. Zacks [10] pointed out that the typical cerebellopontile angle syndrome occurs after a large percentage of head injuries and that hearing, unless of the conductive type, shows abrupt drop for the higher tones.[12] Koch,[13] on reviewing the cases of 146

7. Barbier, J., and Mayoux, R.: A Cerebellar Tumor in the Region of the Vermis, Diagnosed by Vestibular Examination, Rev. d'oto-neuro-opht. **11**:518 (July-Aug.) 1933; abstracted, Arch. Otolaryng. **19**:739 (June) 1934.

8. Brunner, H.: Influence of Cortical Innervation on Labyrinthine Nystagmus, Monatschr. f. Ohrenh. **70**:40 (Jan.) 1936; abstracted, Arch. Otolaryng. **25**:591 (May) 1937.

9. Leidler, R.: What Conclusions Can Be Reached Through a Vestibular Examination as to the Type and Location of an Intracranial Disease? Pract. oto-rhino-laryng. **2**:86 (May); 152 (July) 1939; abstracted, Arch. Otolaryng. **31**:1006 (June) 1940.

10. Zacks, M. A.: Importance of Vestibular Findings Following Injury to the Head, Arch. Otolaryng. **30**:601 (Oct.) 1939.

11. Barré, J. A.: Topographic Syndromes of the Central Vestibular Pathways in Man, Rev. d' oto-neuro-opht. **16**:420 (June) 1938; abstracted, Arch. Otolaryng. **31**:203 (Jan.) 1940.

12. Fowler, E. P., Jr.: Medicine of the Ear, New York, Thomas Nelson & Sons, 1939, p. 349.

13. Koch, J.: Disturbances of Labyrinthine Function After Trauma of the Skull with and Without Injury of the Temporal Bone, Arch. f. Ohren-, Nasen- u. Kehlkopfh. **137**:105, 19399; abstracted, Arch. Otolaryng. **20**:108 (July) 1934.

patients with cranial trauma observed that disturbed function of the inner ear can be caused by cerebral concussion as well as by cranial fractures. Glaser,[14] reported 66 patients in whom he could find no distinct vestibular complex, the only variation from normal being a subnormal response throughout. On the other hand, Portmann and Despons [15] reported 30 cases; they found hyperexcitability in 20, normal responses in 9 and hypoirritability in 7; they stated also that cochlear trouble rarely appears unless the petrous portion of the temporal bone has been fractured.

Comparison of these reports is disheartening. However, they represent all stages of progress for each condition. There is no question but that vestibular findings change with changing progression of each intracranial lesion. Nevertheless, it serves to show that there is still much confusion in the field.

When an otologist differentiates early between a peripheral and a central lesion and designates whether a central lesion is supratentorial or infratentorial, he should feel satisfied, particularly if he does this before suffieient pressure has developed to affect the nuclei in the brain stem and the various cranial centers or caused disturbed equilibration from the cerebellum.

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14. Glaser, M. A.: The Cause of Dizziness in Head Injuries, Ann. Otol., Rhin. & Laryng. **46**:387 (June) 1937.

15. Portmann, G., and Despons, J.: Vestibular Troubles Associated with Cranial Trauma, Rev. de laryng. **58**:585 (June) 1937; abstracted, Ann. Otol., Rhin. & Laryng. **46**:1147 (Dec.) 1937.

SEPTICEMIA DUE TO FRIEDLÄNDER'S BACILLUS

REPORT OF A CASE FOLLOWING CHRONIC OTITIS MEDIA COMPLICATED BY SINUS THROMBOSIS; RECOVERY FOLLOWING THERAPY WITH SULFANILAMIDE AND ITS DERIVATIVES

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There are relatively few cases of infection due to Friedländer's bacillus (Bacillus mucosus capsulatus) to be found in the American literature, and a review of the reports of this infection shows some variance in the actual number of cases.

Baehr, Shwartzman and Greenspan [1] reported 198 cases of Friedländer bacillus infections of all kinds. Of these, only 9 were in the field of otorhinolaryngology. Of these 9 cases, 3 were of acute purulent otitis media, whereas only 1 was of the chronic type. They noted, probably significantly, that in ozena and rhinoscleroma the Friedländer bacillus is a secondary invader, probably introduced by picking the nose after contamination of the fingers, since the organism is common in the gastrointestinal tract of normal persons.

The rarity of infection due to this organism is indicated by Hartman,[2] who stated that only 284 cases of genuine Friedländer bacillus pneumonia have been reported in the medical literature from 1882 to 1938. In turn, it is to be expected that cases of septicemia due to the Friedländer bacillus are also relatively rare. Most reports of this type of infection are to be found in the French literature. Colombe,[3] in 1917, collected 60 cases from the world's literature, in 25 of which the diagnosis was made by a positive blood culture during life.

Caussade, Joltrain and Surmont [4] added 15 new cases which occurred from 1917 to 1924 and reported their own case. Lereboullet and

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1. Baehr, G.; Shwartzman, G., and Greenspan, E.: Bacillus Friedländer Infections, Ann. Int. Med. **10**:1788-1801 (June) 1937.

2. Hartman, M. M.: Recovery from Acute Friedländer's Pneumonia, Ann. Int. Med. **14**:513-521 (Sept.) 1940.

3. Colombe, J.: Les septicémies pneumobacillaires, Thesis, Paris, no. 29, 1917.

4. Caussade, G.; Joltrain, E., and Surmont, J.: Septicémie à pneumobacille de Friedländer, Bull. et mém. Soc. méd. d. hôp. de Paris **48**:148-160 (Feb. 8) 1924.

Denoyelle [5] recorded 2 cases several weeks after the report of Caussade, Joltrain and Surmont, in 1924. Since then a case has been reported by Creyx [6] in 1926 and another by Lereboullet and Pierrot [7] in 1927. In 1928, 2 additional cases were recorded by Brain and Valentine [8] and Mason and Beattie,[9] respectively. Apparently, until 1928 there were only a few cases of septicemia due to Friedländer's bacillus in which recovery occurred. In 1933 Baehr, Shwartzman and Greenspan [10] cited 16 cases of bacteremia due to Friedländer's bacillus, with recovery in 4 cases. In March 1930 Abrami and Worms [11] and in August 1930 Malis [12] reported 2 more cases. From 1934 to 1939 approximately 14 more cases of this type of septicemia were reported.[13] On the other

5. Lereboullet, P., and Denoyelle, L.: Deux cas de septicémies graves à pneumobacilles de Friedländer terminées par la guérison, Bull. et mém. Soc. med. d. hôp. de Paris **48**:226-233 (Feb. 29) 1924.

6. Creyx, M.: Sur un cas de pneumobacillémie, Compt. rend. Soc. de biol. **94**: 596-597 (March) 1926.

7. Lereboullet, P., and Pierrot, M.: Un nouveau cas de septicémie à pneumobacilles de Friedländer terminée par la guérison, Bull. et mém. Soc. méd. d. hôp. de Paris **51**:128-130 (Feb. 10) 1927.

8. Brain, W. R., and Valentine, F. C. O.: Case of Infection of the Meninges and the Blood Stream by Bacillus Mucosus Capsulatus, Lancet **1**:855-856 (April 28) 1928.

9. Mason, E. H., and Beattie, W. W.: Septicemia Due to a Strain of the Bacillus Mucosus Capsulatus Group in a Case of Diabetes Mellitus, Arch. Int. Med. **42**:333-337 (Sept.) 1928.

10. Baehr, G.; Shwartzman, G., and Greenspan, E. B.: Role of Friedländer's Bacillus in Infections, Tr. A. Am. Physicians **48**:353-354, 1933.

11. Abrami, P., and Worms, R.: Septicémie à pneumobacilles de Friedländer avec localisation pulmonaire, Bull. et mém. Soc. méd. d. hôp. de Paris **54**:479-486 (March 31) 1930.

12. Malis, S.: Pneumobacillus Meningitis Secondary to Otitis Media, Arch. Otolaryng. **12**:162-174 (Aug.) 1930.

13. (*a*) Railliet, Pérono and Morel: Septicémie à pneumobacilles avec méningite, Bull. et mém. Soc. méd. d. hôp. de Paris **50**:1693-1695 (Dec. 24) 1934. (*b*) Allen, B.: Otitic Cavernous Sinus Phlebitis Due to Bacillus Friedländer, J. Mt. Sinai Hosp. **2**:169-172 (Nov.-Dec.) 1935. (*c*) Hepp, J.: Gangrène utérine et septicémie à Friedländer post abortum, Ann. d'anat. path. **13**:116-121 (Jan.) 1936. (*d*) Germain, A., and Maudet, J.: Septico-pyohémie à pneumobacille de Friedländer à localisation genito-urinaire initiale et a essaimages multiples, Bull. et mém. Soc. méd. d. hôp. de Paris **52**:1253-1259 (July 20) 1936. (*e*) Laroche, G., and Brocard, H.: Septicémie à pneumobacilles de Friedländer. Mort avec azotémie survenue à la suite d'injections de gonacrine, ibid. **53**:567-569 (May 3) 1937. (*f*) Brunetti, F., Jr.: Setticopiemia de Klebsiella pneumoniae (Friedländer), Gior. di batteriol. e immunol. **18**:91-101 (Jan.) 1937. (*g*) Pfeiffer, D. B.: Klebsiella Pneumoniae (Bacillus Mucosus Capsulatus) Bacteremia Due to Prostatic Abscess: Case with Recovery, Ann. Surg. **106**:1115-1118 (Dec.) 1937. (*h*)

(Footnote continued on next page)

hand, Brodny [14] stated that a review of the literature to 1934 revealed only 51 cases of septicemia due to B. mucosus capsulatus, in only 3 of which the patient recovered.

BACTERIOLOGY

Malis [12] thoroughly reviewed the bacteriologic aspects of Friedländer's bacillus. This organism, the pneumobacillus, belongs to the Bacillus mucosus group and is present as a secondary invader in 5 to 10 per cent of cases of pneumonia. It is gram-negative, as are the other bacteria of the same group. By the precipitation method of von Eisler and Parges it is differentiated from the bacilli found in cases of ozena and rhinoscleroma, which belong to the same group and which are also gram-negative and encapsulated. The other gram-negative, encapsulated bacilli are Bacillus lactis aerogenes of Escherich and Bacillus acidi lactici of Huepse. The former ferments dextrose, saccharose and lactose; the latter, dextrose and saccharose but not lactose. The pneumobacillus may be single, diploform or in short chains. It is nonmotile, possesses no flagella and forms no spores. This bacillus is characteristically surrounded by a well developed capsule. It is easily differentiated from Bacillus coli and various paratyphoid bacilli. The pneumobacillus must also be differentiated from the Pneumococcus mucosus, or the type III pneumococcus, with which it is often confused. The latter differs in that it is a coccus, is gram positive and is soluble in bile.

Julianelle,[15] by agglutination and precipitation tests, was able to distinguish three types of the Friedländer bacillus. Type A, by far the most frequent, and types B and C comprised most of the clinically

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Bonciu, C.: Considération sur deux cas de septicémies dues au bacille de Friedländer, Arch. roumaines de path. expér. et de microbiol. **10**:307-323 (Sept.) 1937. (*i*) Mamone, M., and Perez Fernandez, J.: Sobre un caso de septicemia post-aborto a bacilo capsulado typo Friedländer, Rev. sud.-am. de endocrinol. **20**:125-131 (March) 1937. (*j*) Brulé, M.; Hillemand, P., and Gaube, R.: Septicémie à pneumobacille de Friedländer, Bull. et mém. Soc. méd. d. hôp. de Paris **53**:362-365 (March 22) 1937. (*k*) Giroux, M.: Septicémie à Friedländer et préavitaminose C, Laval méd. **3**:163-166 (May) 1938. (*l*) Jobin, J. B., and Paquet, B.: Septicémie à Friedländer, ibid. **3**:151-162 (May) 1938. (*m*) Septicemia, Bacillus Mucosus Capsulatus, Cabot Case 25251, New England J. Med. **220**:1042-1044 (June 22) 1939. (*n*) Meyer, K., and Amtman, L.: Treatment of Friedländer's Septicemia by Sulfapyridine with Recovery, J. A. M. A. **113**:1641-1642 (Oct. 28) 1939.

14. Brodny, M.: Fatal Case of Septicemia, New England J. Med. **210**:346-348 (Feb. 15) 1934.

15. Julianelle, L. A.: A Biological Classification of Encapsulatus Pneumoniae, J. Exper. Med. **44**:113-128 (July) 1926; Immunological Relationships of Encapsulated and Capsule-Free Strains of Encapsulatus Pneumoniae (Friedländer's Bacillus), ibid. **44**:683-696 (Nov.) 1926; The Distribution of Friedländer's Bacilli of Different Types, ibid. **52**:539-545 (Oct.) 1930.

important strains. All the other strains fell into a heterogenous group, X. Julianelle pointed out that the Friedländer bacillus has been reported as occurring in the upper respiratory tract of normal people in an incidence of 0.5 to 8 per cent.

The first instance of cure of septicemia due to the Friedländer bacillus was reported by Abrami and Worms in 1930[11]; in this case the therapeutic agent was acriflavine hydrochloride, injected by vein. Apparently, no other cases have been reported in which this drug was used.

With the advent of sulfanilamide and its derivatives, it was natural that these drugs should be tried in treatment of infections of this sort. Early animal experimentation was discouraging. Gross, Cooper and Lewis[16] verified observations by other investigators on the doubtful value of sulfanilamide therapy of Friedländer bacillus infections in mice and concluded that the efficacy of sulfanilamide and its derivatives in treatment of such infections in mice was still open to question. They could find no therapeutic effect of sulfanilamide in mice infected with less than 10 minimal lethal doses.

Nevertheless, only one month previous to the last-mentioned report Brown[17] described a case of abscess of the lung of seventeen years' duration which was cured with sulfanilamide. The following year, 1939, Meyer and Amtman[13n] reported their case of Friedländer bacillus septicemia in which recovery followed treatment with ordinary doses of sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine). Solomon[18] stated that on the basis of the results of treatment in 17 cases of chronic infection of the lung the use of sulfanilamide and its derivatives was warranted.

REPORT OF CASE

F. P., a 58 year old white man, was first admitted on June 13, 1938 to the surgical service of Dr. Donald G. Glover, St. Luke's Hospital, Cleveland, complaining of swelling of the left side of the neck and the left zygomatic area for four weeks and otalgia and otorrhea on the same side for fifteen to twenty years. Examination revealed a poorly nourished man, who was not acutely ill. The findings were consistent with the diagnosis of chronic mastoiditis and subacute cervical abscess on the left side and subcutaneous abscess over the left zygoma. The patient was transferred to the nose, ear and throat service, and on June 15 a simple mastoidectomy was performed on the affected side. The plate of the lateral sinus was

16. Gross, P.; Cooper, F. B., and Lewis, M.: Sulfanilamide Therapy of Friedländer's Bacillus Infections of Mice, Proc. Soc. Exper. Biol. & Med. **39**:12-13 (Oct.) 1938.

17. Brown, P. K.: Sulfanilamide: A Specific for the Friedländer Bacillus, California & West. Med. **49**:144 (Aug.) 1938.

18. Solomon, S.: Chronic Friedländer Infections of the Lungs: Report on Seventeen Cases and Observations on Therapy with Sulfapyridine and Sulfanilamide, J. A. M. A. **115**:1527-1536 (Nov. 2) 1940.

observed to be necrotic, and on removal a considerable amount of free pus escaped. It was found that the abscess had dissected along the jugular vein and had localized in the upper cervical region, forming the cervical abscess. In addition, a tract was seen leading to the zygomatic abscess. Unhealthy granulations were observed over the lateral sinus but were not disturbed. Drainage of both the zygomatic and the cervical abscesses was effected. Cultures of material from the mastoid process and the cervical abscess were typical for B. mucosus capsulatus.

Five days after this procedure the patient began to have a spiking temperature, and a blood culture was positive for Friedländer's bacillus. This result was substantiated on the following day.

On June 22 (the seventh postoperative day) exploration confirmed the diagnosis of thrombosis of the left lateral sinus. Free bleeding was obtained from the upper end of the sinus. The jugular vein was not ligated. The immediate postoperative course was stormy. The patient received a total of 740 grains (48.1 Gm.) of sulfanilamide and 2,250 cc. of citrated blood in the ensuing seven days. The free sulfanilamide content of the blood ranged from 6.3 to 15.4 mg. per hundred cubic centimeters. Two days after this therapy was instituted, the blood culture became negative for the organism. Previously four blood cultures had been positive for Friedländer's bacillus. On the sixth day after the second surgical procedure the condition had resolved by lysis, and thereafter convalescence was uneventful.

Three weeks later the patient returned to the hospital for a three day period of observation. At this time there was still some drainage from the left ear and the cervical abscess. Blood cultures were negative for Friedländer's bacillus. He received 180 grains (11.7 Gm.) of sulfanilamide, with resultant improvement.

On March 8, 1939 the patient was readmitted to the genitourinary service, complaining chiefly of pain in the perineum. Incision and drainage of a prostatic abscess was carried out; cultures revealed Friedländer's bacillus as the causative organism. In a period of three days 160 grains (10.4 Gm.) of sulfanilamide was administered. On Sept. 23, 1940 another prostatic abscess was evacuated at Lakeside Hospital, and B. mucosus capsulatus was again found. After this he was advised to continue sulfanilamide therapy in moderate doses for three months.

The patient was not seen again until April 12, 1941, when he presented himself with acute suppurative otitis media of the right ear. Friedländer's bacillus was found in the discharge. Sulfanilamide was again given, and on May 3 he was discharged as cured.

SUMMARY

An unusual case of septicemia due to Friedländer's bacillus following chronic purulent otitis media and complicated sinus thrombosis is reported.

Recovery was effected by the use of sulfanilamide.

The literature is reviewed.

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ENDAURAL MASTOIDECTOMY

EXPERIENCE IN A SERIES OF SEVENTY-SIX CASES

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On Dec. 4, 1939, at a meeting of the Chicago Laryngological and Otological Society, we presented our experience with endaural mastoid surgery.[1] At that time 39 endaural procedures were reviewed. The purpose of this report is to review our total experience with this method in our first 76 cases over the past eighteen months. Of these, 29 were cases of complete, or simple, mastoidectomy, 5 of atticomastoidectomy, or modified radical mastoidectomy, 41 of mastoidotympanectomy, or radical mastoidectomy and 1 of the fenestration operation for otosclerosis. Of the 76 cases, 3 occurred in private practice, 12 at the Illinois State Penitentiary, Joliet, Ill., and the remainder at the Illinois Eye and Ear Infirmary, Chicago. A considerable portion of the surgical work reported in this article was done by the resident staff of the Illinois Eye and Ear Infirmary, under our personal supervision, after a course in cadaver technic and surgical demonstrations. No attempt will be made to present the actual technics of the operations except that of atticomastoidectomy, since the other procedures have been fully described in Lempert's [2] presentation.

ENDAURAL COMPLETE MASTOIDECTOMY

For the purpose of simplifying our report we have abstracted in table 1 our records of the first 28 consecutive cases of endaural complete mastoidectomy we performed.

In our experience there is no area of the mastoid which cannot be reached adequately by the endaural approach. Of the cases recorded in table 1, subperiosteal abscess or marked edema of the soft tissues was encountered in 12. This did not prevent the performance of a thorough mastoidectomy, which should be the primary requisite of all mastoid sur-

1. Henner, R.: Experiences with Endaural Complete Mastoidectomy and Atticomastoidectomy, Arch. Otolaryng. **31**:1022-1023 (June) 1940. Woodruff, G.: Observations on the Endaural Mastoidotympanectomy of Lempert, ibid. **31**:1024-1028 (May) 1940.

2. Lempert, J.: Endaural Antauricular Surgical Approach to the Temporal Bone: Principles Involved in this New Approach, Arch. Otolaryng. **27**:555-587 (May) 1938.

TABLE 1.—*Data on Cases of Endaural Complete Mastoidectomy*

Case No.	Name	Age, Yr.	Date of Operation	History	Pathologic Condition	Operative Complications	Postoperative Course	End Results
1	M. P.	24	4/12/39	Otorrhea 4 wk.; tenderness and external swelling	Subperiosteal abscess; coalescent mastoiditis	None	Uneventful	Good
2	A. M.	4	4/19/39	Postauricular fistula following mastoidectomy in 1939	Diseased periantral cells; revision of mastoidectomy	Fistula dissected and everted and closed with purse-string suture	Suture sloughed; wound closed in 2 wk. by second intention	Good
3	V. C.	11	4/28/39	Pain 13 days; paracentesis 3d day; profuse discharge and sagging	Marked involvement of zygomatic, retrosinal and tip cells	None	Temperature 105 F. on 8th postoperative day; horizontal nystagmus to same side; acute pharyngitis; response to sulfanilamide	Good
4	S. S.	8	5/ 2/39	Temperature 104.6 F.; stupor; bilateral otitis media recurrently	Acute zygomatic, extradural abscess with necrosis of squamosa	None	Temperature resolved gradually; course otherwise uneventful	Dry perforation of drum
5	E. V.	12	5/17/39	Pain 10 days; discharge from attic perforation 3 days; temperature 103.6 F.; sagging	Subperiosteal abscess; perisinal abscess	None	Temperature 104 F. for 4 days; sulfanilamide used; temperature normal thereafter	Dry attic perforation
6	D. L.	5	5/24/39	Stormy preoperative course with pain 13 days; myringotomy 4th day; delirium; postauricular edema	Subperiosteal abscess; complete coalescence of mastoid	Dural exposure	Uneventful	Good
7	M. G.	8	5/24/39	Otorrhea 4 wk.; cessation of discharge; sagging 4 days before operation; central perforation	Coalescent mastoiditis	None	Uneventful	Good
8	P. U.	24	5/31/39	Otorrhea 3 mo.; headaches and dizziness; nausea and vomiting 7 days	Coalescent mastoiditis	Lateral sinus injured; packs removed on completion of operation	Uneventful	Good
9	N. P.	12	6/21/39	Otorrhea 2 wk.; postauricular swelling; sagging	Subperiosteal abscess	None	Uneventful	Good
10	C. L.	24	7/15/39	Pain and discharge in 3 wk.; tenderness of tip; nystagmus to same side	Tip abscess; coalescent mastoiditis	None	Nystagmus continued for 4 days; otherwise uneventful	Good
11	W. K.	60	7/19/39	Pain in ear 6 wk.; no discharge; roentgenologic evidence of mastoid coalescence	Necrosis of deep cells	None	One week after patient was up and around bronchopneumonia developed; good response to sulfanilamide	Good

12	N. C.	3	9/24/39	Chronic suppuration with acute exacerbation; postauricular swelling	Subperiosteal abscess; foul pus in antrum	None	Uneventful	Foul discharge from ear (see case 24, table 3)
13	A. P.	20	10/ 4/39	Sagging; marked edema over mastoid	Subperiosteal abscess; tip abscess; perisinal abscess; sinus normal	None	Uneventful	Good
14	A. F.	10	10/25/39	Sagging; postauricular edema; albumin in urine ++++	Retrosinal and retrofacial necrosis	Anterior canal wall injured by incisions	Urine cleared in several days; occlusion of canal by granulations from anterior canal wall; granulations removed; epithelial filings grafted on raw areas	External canal healed, with scarring; no stenosis
15	P. M.	8	11/15/39	Postauricular swelling; discharge 3 wk.	Subperiosteal abscess; tip abscess; fistula in region of digastric muscle; sinus granulations	None	Uneventful	Good
16	M. M.	4	11/22/39	Otitis media 1 mo.; tenderness and pain	Fistula in tip; coalescent mastoid	None	Secondary anemia marked	Good
17	S. C.	9	12/13/39	Active tuberculosis; discharging ear of unknown duration; pain and tenderness of mastoid	Pale, patchy necrosis of cells	Local anesthesia	Uneventful	Good
18	F. L.	32	12/20/39	Foul discharge; acute exacerbation of chronic otitis media; swelling	Subperiosteal abscess; necrosis of tip	None	Uneventful	Good
19	J. S.	14	1/17/40	Otorrhea 3 wk.; postauricular swelling	Abscess of tip and retrosinal necrosis	None	Uneventful	Good
20	J. H.	8	2/ 4/40	Pitting edema; otorrhea 4 wk.	Subperiosteal abscess; sinus granulations	None	Uneventful	Good
21	C. H.	15	2/ 7/40	Headache 3 days; chills and fever; discharge 2 wk.; temperature 102 F.	Retrosinal necrosis and coalescent mastoiditis	None	Uneventful	Good
22	R. S.	6	2/28/40	Tenderness and edema; otorrhea 5 wk.; hemoglobin 50%; red blood cells 2,500,000; 2 preoperative transfusions	Perisinal abscess	None	Uneventful	Good

TABLE 1.—*Data on Cases of Endaural Complete Mastoidectomy—Continued*

Case No.	Name	Age, Yr.	Date of Operation	History	Pathologic Condition	Operative Complications	Postoperative Course	End Results
23	G. H.	4	2/28/40	Acute exacerbation of chronic ear condition; retroauricular swelling of mastoid; intermittent discharge 2 yr.	Patchy sclerosis with granulations in mastoid and zygomatic cells	None	Uneventful	Good
24	W. F.	71	3/13/40	Diabetic acidosis; profuse discharge 3 wk.; sagging; tenderness; diabetes just under control when 1° nystagmus developed	Pus and granulations in antrum; cell over sinus full of pus; tip abscess	Local anesthesia	Uneventful; diabetes controlled	Good; 2 mo. after operation patient died in another hospital of diabetic acidosis
25	L. R.	27	4/ 4/40	Earache and otorrhea 10 days; chronic otitis media; drum gone; patient 3 mo. pregnant, treated elsewhere with sulfanilamide; tenderness in neck and cervical adenitis; tenderness over emissary vein; preoperative temperature 105.2 F.; chill; blood culture negative; red cells 3,000,000; hemoglobin 61%	Sclerotic mastoid with pus under pressure in perisinal area; liquefaction necrosis of sinus from 1½ inches behind upper knee to below bulb; enlarged antrum	Jugular ligation after obtaining free bleeding from above and none from below by suction; 2 transfusions at operation	3d transfusion on 2d postoperative day; no sulfanilamide; uneventful recovery	Good; granulations in middle ear moist (to have tympanectomy later)
26	F. B.	2	5/ 6/40	Foul otorrhea 3 mo.; ceased for 2 days and began again; anterior perforation; postauricular swelling	Fistula in cortex; coalescent mastoid widely pneumatized for 2 yr. old child	None	Uneventful	Good
27	M. S.	10	5/22/40	1937 postauricular mastoidectomy; 1939 Wilde's incision for recurrence of pain and swelling and discharge for 5 days; temperature 102 F.	Sequestrum found over dura; sinus and dura exposed	None	Uneventful	Good
28	A. G.	18	6/21/40	Discharge 1 mo. ago, stopped in few days; whispered voice ad concham; pain 1 mo.; glands over mastoid enlarged; roentgen treatment; paracentesis of beefy drum; no pus	Mucopus in antrum; patchy sclerosis of mastoid with granulations; no breaking down	None	Uneventful	Good

gery. There were 8 cases of tip abscess and 5 of marked retrosinal disease. In our first cases of this type we met with some difficulty in cleaning out the involved areas; however, as we gathered experience this was overcome. It appears to us that the zygomatic cell area is more easily exposed through the endaural than through the postauricular route.

There were 6 cases of perisinal abscess, 1 of thrombosis of the lateral sinus, complicated by pregnancy, 3 of extradural abscess of the middle fossa and 4 of labyrinthitis. Two of the cases of extradural abscess and all those of labyrinthitis are included with the cases of mastoidotympanectomy. A thorough operation was performed in all these cases, with no unusual difficulty. There has been no death or any serious illness in any of these cases. Therefore we feel that the presence of any of the aforementioned complications of mastoiditis is not a contraindication to the use of the endaural approach.

The following advantages of endaural complete mastoidectomy have impressed us in particular.

1. Routine postoperative inspection of the mastoid wound cavity, while not usually necessary, may be attained by merely retracting the edges of the wound with a nasal speculum. There are no stitches to be removed.

2. Packing is unnecessary in the endaural operation; thus continuous open drainage is afforded. Postoperative complications appear to be somewhat less severe and less frequent since we have adopted this method in the clinic. A properly performed operation leaves an open window with unimpeded drainage. It is, of course, possible to provide wide open drainage with the postauricular method, but only by leaving the wound wide open and unsutured, thus risking delayed healing and fistula formation. The predominant practice in this country is to close the wound more or less completely and to introduce a drain or pack into the cavity through the unsutured portion of the skin wound. This usually does not give as free drainage as does the open, unimpeded endaural window. This point has been thoroughly discussed by Lempert.[2]

3. The postoperative care is more simple than that following the postauricular operation. In the majority of cases the bandage is discarded at the end of four days, and after that the only dressing needed is a piece of sterile cotton in the external canal, which can be changed by the patient when it becomes soiled. Occasionally granulations have to be cauterized, and in a few cases the opening of the wound may need to be lightly spread with a nasal speculum to keep it from closing too soon.

4. Pain and stiffness of the neck from injury to the attachment of the sternocleidomastoid muscle occur less frequently than with the postauricular method.

5. It is impossible for a retroauricular fistula to form after the endaural operation. Healing may be delayed, though it seems to occur less frequently than after the postauricular operation. We had 1 case in which the wound healed very slowly, but after a few months it closed, leaving only an inconspicuous and harmless depression in the posterior canal wall. It seems probable that employment of the postauricular method in this case would have resulted in a deep, unsightly depression or in a postauricular fistula.

6. In our opinion the cósmetic result is of secondary importance, but in general the endaural method gives a decidedly better result than the postauricular one.

The main disadvantage of the endaural approach for complete mastoidectomy is the necessity of performing the mastoid exenteration through a relatively small opening, which affords a less comprehensive view of the bony structure in the various stages of the operation than does use of the postauricular route. By a less comprehensive view we mean that all areas cannot be seen at one and the same time.

In trying to determine which is the better method of performing a complete mastoidectomy, the advantages of one method must be weighed against those of the other. It is perhaps unfortunate that after performing this work together we are not in complete agreement as to the method of choice for complete (simple) mastoidectomy. Because this slight difference of opinion exists it seems essential to give in brief the background of experience of each of us.

One of us, G. H. W., has had about nineteen years' experience in mastoid surgery. All of this work until the past two years was done by the postauricular route. Since then nearly all operations have been done endaurally. In addition, 15 to 20 operations have been performed on cadavers, part of them during a week's visit at Dr. Lempert's clinic and under his guidance.

The other, R. H., has had about seven years' experience in mastoid surgery, and for the past two years all the work has been done endaurally. In addition, about 50 to 60 operations have been carried out on cadavers, many of them during a six week's course under Dr. Lempert's guidance.

It will be seen that one of us, G. H. W., had a fairly long and extensive experience with postauricular surgery before taking up the endaural method. On the other hand, the other, R. H., had a rather short and limited experience with postauricular surgery before his extensive endaural practice of the past two years.

The endaural work on the living has been about equally divided between us. It may be that this difference in surgical experience accounts in some measure for the slightly different conclusions we have formed concerning the endaural complete mastoidectomy.

Conclusions.—R. H. feels that the endaural route is the method of choice in complete mastoidectomy for the following reasons:

1. Since acquiring experience in the method there has been no difficulty in adequately handling any type of pathologic condition in any area of the mastoid.

2. The after-care is simplified.

3. The minimal trauma to normal tissues and the maintenance of wide open drainage appear to lessen postoperative complications.

It is a fact that the endaural approach limits the field of operation for the inexperienced operator and for the demonstration of surgical technic. However, the aforementioned advantages outweigh this limitation.

G. H. W. feels that the question of the choice of method roughly boils down to this: The complete mastoid operation is more difficult to perform by the endaural than by the postauricular route. On the other hand, the period of convalescence following the endaural operation is usually easier for the patient and simpler for the surgeon than that following a postauricular operation. The details have already been discussed. After careful consideration based mainly on his own experience, the following conclusions are reached:

1. Proficiency in the endaural complete mastoidectomy is gained only after thorough study of the method, followed by carefully performing at least ten to twelve such operations on the cadaver.

2. A surgeon thoroughly proficient in the endaural method can adequately carry out all steps of the complete mastoid operation and is then justified in employing the method, thereby securing the advantages of a more favorable postoperative course.

3. A surgeon who has not acquired this proficiency should not attempt to use the endaural route for complete mastoidectomy. The operation in his hands is not a safe procedure. He may easily do incomplete work or injure important structures.

ATTICOMASTOIDECTOMY

Atticomastoidectomy is reserved for those cases in which a chronically discharging ear is associated with good hearing. This approach is used particularly in cases of necrosis and perforation of the attic with an intact pars tensa in order to preserve the hearing. The technic of the operation is as follows:

1. After the performance of a complete mastoidectomy the dural plate is skeletonized forward until the upper portions of the malleus are well exposed. This may involve the removal of cholesteatoma or other attic disease.

TABLE 2.—*Data on Cases of Atticomastoidectomy*

Case No.	Name	Age, Yr.	Date of Operation	History	Pathologic Changes	Operative Complications	Postoperative Course	End Results
1	F. W.	31	8/17/39	Otorrhea intermittent for 2 yr.; attic perforation; whispered voice, 10 ft. (304 cm.)	Attic and antral cholesteatomas; sclerotic mastoid	None	Uneventful	Dry ear; whispered voice heard at 20 ft. (608 cm.)
2	A. L.	23	11/ 1/39	Otorrhea 6 mo.; previous attack 11 yr. ago; attic perforation; whispered voice, 6 inches (15 cm.)	Few isolated diseased cells; pus in tip; antrum enlarged	None	Uneventful	Dry ear; whispered voice, 2 ft.
3	R. L.	65	12/27/39	Otorrhea 3 mo.; large attic perforation; polyp in attic; spoken voice, contact	Sclerotic mastoid; granulations in antrum and attic	None	Uneventful	Dry ear; spoken voice, 3 ft. (91 cm.)
4	R. T.	29	6/13/40	Otorrhea for many years; whispered voice, 2 ft. (61 cm.); central perforation	Necrosis in body of mastoid; sinus far forward	Sinus accidentally opened by burr; bleeding easily controlled	Uneventful	Slight moisture at times; whispered voice, 2 ft.
5	H. M.	51	6/12/40	Otorrhea for many years; 3 attacks of vertigo with nystagmus; central perforation; whispered voice, 15 ft. (457 cm.)	Attic necrosis with periantral purulent material; hypotympanic granulations curetted through perforation without disturbing drum	None	Uneventful	Dry ear, with inferior perforation; whispered voice, 15 ft.

2. The posterior, superior and anterosuperior canal walls, including the tympanic annulus, are thinned and finally removed, without disturbing the continuous membranous canal wall and the drum membrane.

3. If the incus is present, it may be removed if it seems advisable. Removal of the head of the malleus for mobilization of the flap is not always necessary here, as it is in the fenestration operation.

Fig. 1.—Endaural incisions delineated in the antauricular suprameatal membranous triangle and the creation of a mobile endaural membranous and extracartilaginous window for the performance of a mastoidectomy or an attico-mastoidectomy.

4. The flap is cut as in Lempert's fenestration operation. This is done when possible so as to exclude the attic perforation. In 3 of our cases atticotomy was performed without cutting a flap.

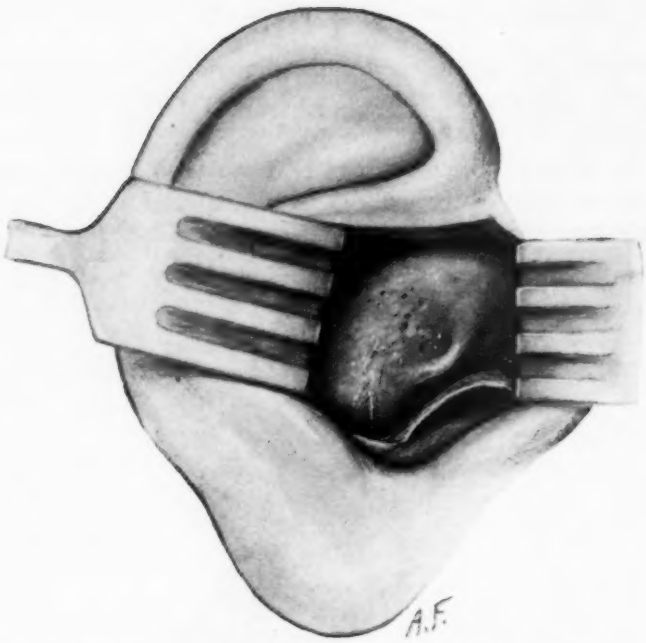

Fig. 2.—Completed antauricular exposure of the mastoid process.

Fig. 3.—Complete mastoidectomy, with sharp definition of the lateral sinus plate, dural plate and external semicircular canal.

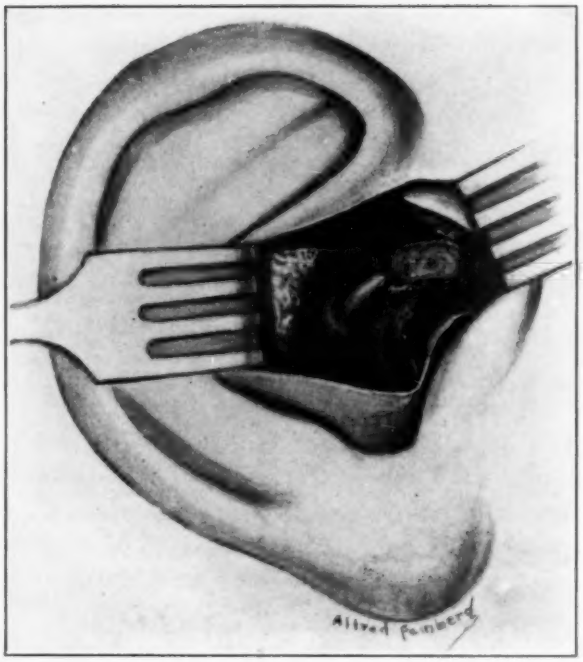

Fig. 4.—Exposure of the dural plate carried forward, showing cholesteatoma in the attic.

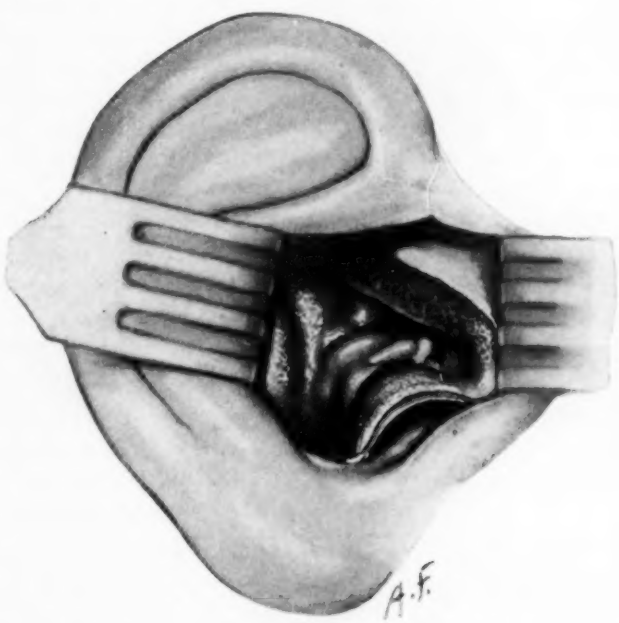

Fig. 5.—Skeletonization of the anterosuperior bony wall of the external auditory canal, resulting in the exposure of the entire epitympanic recess, allowing for the removal of cholesteatoma, granulation and diseased ossicles in the attic.

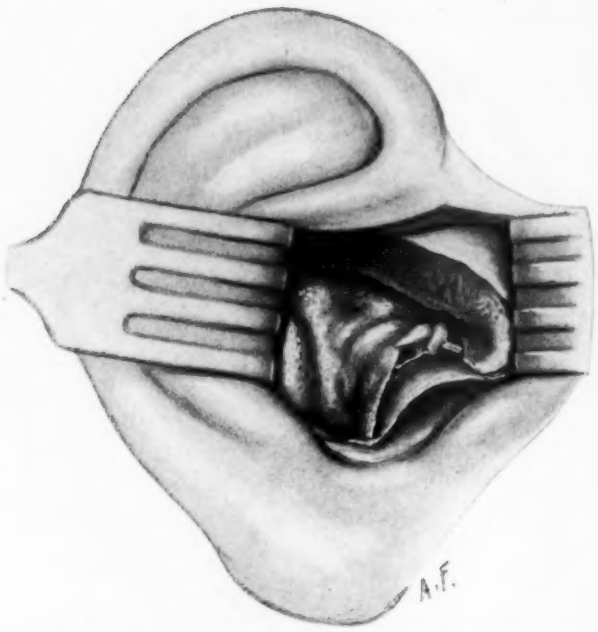

Fig. 6.—Posterior, superior and anterosuperior bony canal wall removed to and including the sulcus tympanicus so as to liberate the tympanomeatal cutaneous membrane.

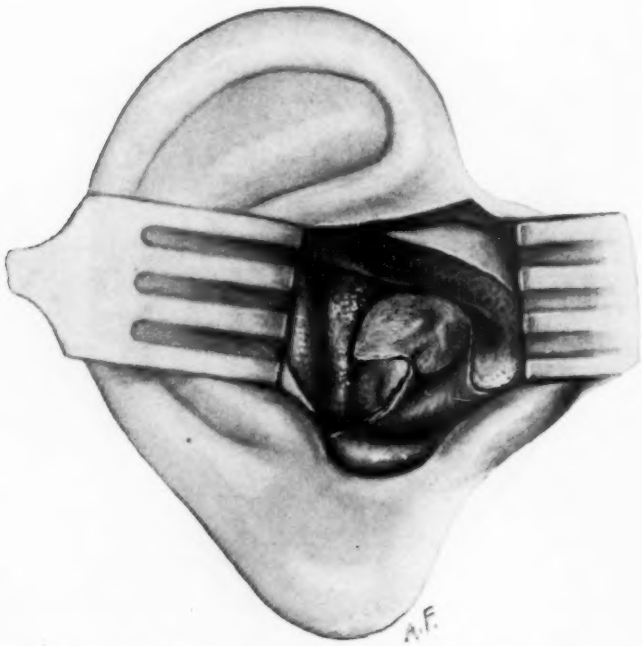

Fig. 7.—Liberated but completely intact tympanomeatal cutaneous membrane rotated posteriorly and placed in position so as to seal off the tympanic cavity.

5. The flap is rotated posteriorly into the mastoid cavity and tensed in place with paraffin mesh.

In our 5 cases we have had a completely uneventful postoperative course. This operation, when applicable, allows for the complete removal of diseased tissue from the mastoid and attic with preservation of, and in some cases even improvement in, hearing.

Conclusions.—This is the ideal approach in cases of pathologic conditions of the attic in the presence of serviceable hearing for the following reasons:

1. The exposure brings one directly over the attic, facilitating the minute dissection necessary in removing the diseased tissue about the ossicles.

2. The flap is continuous with the drum membrane; thus in many cases the middle ear is again sealed off, which affords improved hearing.

3. To those interested in fenestration surgery this operation offers the most valuable experience with the living, since the technic is the same except for not creating the fenestra.

MASTOIDOTYMPANECTOMY

Table 3 summarizes the essential histories in 41 cases of mastoidotympanectomy. Our indication for this operation was persistent foul suppuration in spite of prolonged conservative treatment, including attic irrigation and polypectomy, plus the loss of serviceable hearing. Other indications for operation were headache and dizziness, of true vertiginous type, with or without a positive fistula test, actual labyrinthitis and threatening intracranial complications. In the 3 cases of acute labyrinthitis we were able to follow conservative methods for from two to four weeks, until the active labyrinthitis subsided, and then we undertook surgical treatment.

Without discussing the actual technical procedures, stress should be laid on the management of lesions in the middle ear in order to obtain a dry ear. Both general and local anesthesia have been used. The former is preferable unless contraindicated. The direct view afforded endaurally makes it possible to perform operations with meticulous care on all parts of the tympanic cavity. We agree thoroughly with Lempert that once a radical operation is undertaken complete tympanectomy should be performed. This should include a complete mastoidectomy when indicated plus the following procedures:

1. Thorough removal of diseased tissue from all parts of the attic, with complete removal of the lateral wall of the attic, the incus and the malleus.

2. The use of cotton plugs soaked with epinephrine to strip the middle ear of all mucosa and remnants of tympanic membrane. This procedure allows the operator to work in a comparatively dry field.

TABLE 3.—*Data on Cases of Endaural Mastoidotympanectomy*

Case No.	Name	Age, Yr.	Date of Operation	History	Pathologic Changes	Operative Complications	Postoperative Course	End Results
1	A. J.	21	11/ 8/38	Otorrhea for many years; conservative treatment for 3 mo.	Enlarged antrum; granulations and periantral necrosis	None	Uneventful	Dry ear
2	M. K.	57	11/ 9/38	Left simple mastoidectomy 4 yr. ago; persistent otorrhea since; dizziness	Granulations in middle ear; facial twitching on manipulating granulations; sclerosed mastoid	None	Surgical erysipelas 10th postoperative day	Dry ear
3	I. A.	47	11/17/38	Headaches and dizziness; otorrhea for many years	Granulations and polyps in middle ear and antrum; desquamated material in attic	None	Uneventful	Dry ear; canal somewhat shallowed
4	E. B.	38	12/31/38	Otitis media 2 yr. ago; otorrhea 3 wk.; polyp blocking canal; 2° nystagmus; dizziness; bed rest 16 days before operation; turning test showed diminished vestibular function; hearing ad concham	Large cholesteatoma seen with dural exposure; fistula in posterior canal wall; granulations on horizontal canal and over facial nerve	None	Uneventful recovery	Dry ear; no further labyrinthine symptoms
5	A. A.	13	1/ 4/39	Otorrhea 12 yr.	Granulations and bone necrosis in middle ear	None	Uneventful	Dry ear
6	J. Z.	32	4/20/39	Otorrhea for many years; headaches	Sclerotic mastoid, with granulations in middle ear	None	Uneventful	Dry ear
7	F. L.	40	4/16/39 12/17/39	Foul otorrhea for 22 yr.; headaches; dizziness; falling to the same side while walking; polypectomy in 1930; simple mastoidectomy in 1936	Few necrotic periantral cells; middle ear obliterated by granulations	None	Dry for 7 mo.; when posterior part of radical cavity began to bulge; roentgenograms revealed retrosinal cells fused into a cystlike cavity; cyst full of gelatinous material, removed endaurally	Dry ear; no further labyrinthine symptoms
8	M. G.	19	5/10/39	Otorrhea for 7 yr.; drum gone	Cholesteatoma in enlarged antrum	None	Uneventful recovery	Dry ear
9	J. H.	23	6/ 7/39	Scarlet fever, followed by otorrhea for 16 yr.; dizziness; radical mastoidectomy in 1928; headaches; persistent discharge	Antrum containing mucoid material; zygomatic cells found	None	Uneventful	Dry ear
10	J. N.	33	6/13/39	Otorrhea 29 yr.; central perforation; granulations treated with trichloroacetic acid and removed several times	Sclerotic mastoid; polyp in middle ear	None	Uneventful	Dry ear
11	P. T.	37	6/14/39	Otorrhea 25 yr.; 4 polypectomies; polyp present	Polyp in antrum; few periantral cells; softened area in tegmen tympani	Accidental perforation of lateral sinus	Uneventful; no pack left on sinus after hemorrhage was controlled	Mucoid moisture from middle ear
12	M. O.	4	6/21/39	Bilateral otorrhea following scarlet fever; poorer-hearing ear operated on	Sclerotic left mastoid; enlarged antrum with granulations	None	Uneventful	Slight mucoid moisture from cell behind facial ridge

13	T. L.	30	6/21/39	Foul discharge for 3 yr.; vertigo at times	Antrum enlarged; ossicles carious	Sinus plate and dural plate removed	Uneventful	Patient left clinic before treatments were completed
14	R. M.	20	6/28/39	Otorrhea for 2 yr.; headaches and dizziness for 2 mo.; large central perforation	Few necrotic cells; enlarged antrum; cholesteatoma	None	Uneventful	Dry ear
15	J. K.	62	7/ 5/39	Otorrhea for 8 yr.; marginal perforation	Sclerotic mastoid; antral cholesteatoma	None	Uneventful	Dry ear; shallow canal
16	J. Z.	21	7/26/39	Mucoid otorrhea for 10 yr.; poor hearing	Enlarged antrum, full of purulent material; granulations of middle ear	None	Uneventful	Dry ear
17	A. S.	14	8/ 4/39	Bilateral otorrhea 10 yr.; polyp in attic	Sclerotic mastoid; cholesteatoma	Endosteum exposed over horizontal canal; facial twitchings	Facial weakness for 10 days; complete recovery	Dry ear
18	F. R.	13	8/30/39	Foul otorrhea for 10 yr.; headache and dizziness; attic perforation	Cellular mastoid with necrotic coalescence; attic necrosis with ossicular caries	None	Uneventful	Dry ear
19	C. R.	56	9/13/39	Otorrhea 6 mo.; pulsating inferior marginal perforation	Antrum much enlarged; many cells with pus; granulations in middle ear; peritubal bone necrosis	None	Uneventful	Dry ear
20	W. D.	42	9/20/39	Intermittent otorrhea since childhood; drum gone; granulations	Cholesteatoma; sclerotic mastoid	None	Uneventful	Dry ear
21	F. W.	18	12/14/39	Scarlet fever in 1928; simple mastoidectomy in 1928; modified radical mastoidectomy in 1931;(foul discharge; stenotic canal; postauricular fistula	Granulations in middle ear; hypotympanic granulations and bone necrosis	None	So-called scarlet fever, isolation at another hospital	Dry ear, with small canal
22	E. H.	37	12/20/39	Otorrhea for 30 yr. after scarlet fever; dizzy spells, lasting several minutes	Sclerotic mastoid; granulations in antrum and middle ear	None	Uneventful	Dry ear
23	R. B.	38	1/11/40	Otorrhea for many years; headaches and dizziness	Polypoid granulations; active bone absorption over sinus	None	Uneventful	Dry ear
24	N. C.	3	1/24/40	Chronic otorrhea since scarlet fever at age of 2; previous endaural simple mastoidectomy (see case 12, table 1)	Sequestrum of common crus of superior and posterior vertical canals	None	Uneventful	Moisture; labyrinthine operation to be performed
25	R. F.	28	1/31/40	Foul otorrhea since 5 yr. of age; inferior perforation	Granulations in middle ear; no ossicles seen	Facial nerve exposed; no paralysis	Uneventful	Dry ear
26	S. S.	34	2/18/40	Otorrhea for 26 yr.; headaches and dizziness; polyp in canal	Ossicular caries and polypoid granulations in sclerotic mastoid	Stapes extracted in removing granulations at processus cochleariformis	Uneventful	Dry ear
27	W. F.	34	2/22/40	Otorrhea for 17 yr.	Sclerotic mastoid; enlarged antrum	None	Uneventful	Dry ear

TABLE 3.—*Data on Cases of Endaural Mastoidotympanectomy—Continued*

Case No.	Name	Age, Yr.	Date of Operation	History	Pathologic Changes	Operative Complications	Postoperative Course	End Results
28	J. C.	33	3/14/40	Otorrhea for 3 yr.; dead labyrinth following acute suppurative labyrinthitis	Sclerotic mastoid; granulations on the horizontal canal	None	Uneventful	Dry ear
29	C. W.	32	4/ 4/40	Otorrhea for 20 yr.; headaches; vertigo	Sclerotic mastoid; caries of ossicles	None	Uneventful	Dry ear
30	J. M.	42	4/ 5/40	Acute suppurative manifest labyrinthitis; total deafness; third degree nystagmus; no caloric response; bed rest 4 wk. before operation; otorrhea 4 yr.	Sclerotic mastoid; cholesteatoma; fistula of horizontal canal	None	Uneventful	Dry ear
31	V. W.	32	4/18/40	Otorrhea for many years; headaches; dizziness	Diseased periantral cells; mucopus; polyp in middle ear	None	Uneventful	Dry ear
32	A. G.	18	4/24/40	Otorrhea for years; radical mastoidectomy (postauricular) in 1938 for acute exacerbation	Hypotympanic granulations; caries of tegmen antri	None	Uneventful	Dry ear
33	T. S.	35	5/ 2/40	Otorrhea for years; headaches; dizziness; large central perforation	Sclerotic mastoid; forward lying sinus; hypotympanic necrosis	Jugular bulb perforated in cleaning out hypotympanum; bleeding controlled by pack	Uneventful	Dry ear
34	T. N.	14	5/ 8/40	Foul otorrhea 3 yr.; attacks of dizziness; large central perforation	Granulations in attic and tympanum	None	Uneventful	Dry ear
35	M. R.	27	5/15/40	Foul otorrhea 13 yr.; dizzy spells; attic perforation	Sclerotic mastoid; enlarged antrum; ossicular caries	None	Uneventful	Dry ear
36	B. W.	12	5/15/40	Otorrhea 10 yr.	Sclerotic mastoid; tympanum filled with granulations	None	Uneventful	Dry ear
37	L. N.	10	5/22/40	Foul otorrhea 6 yr.; large perforation; polyp in canal	Sclerotic mastoid; granulations in antrum and attic	None	Uneventful	Dry ear
38	E. B.	14	6/ 5/40	Otorrhea for many years; polyp removed in 1939	Antrum filled with granulations; diseased cells in tip	Jugular bulb exposed; no hemorrhage	Uneventful	Dry ear
39	J. G.	20	6/ 5/40	Otorrhea 17 yr.; middle ear filled with granulations	Broken-down mastoid; large tip abscess; granulations over lateral sinus	None	Uneventful	Dry ear
40	J. D.	..	6/12/40	Otorrhea many years; headaches; progressive rise in temperature past few days: 101, 102, 103 and 103.6 F.	Body of mastoid sclerotic; sinus plate normal; extradural abscess of middle fossa	None	Low fever for 2 wk.; course then uneventful	Referred out of town for after-care; no report
41	F. K.	..	6/19/40	Intermittent otorrhea 15 yr.; occasional headaches	Sclerotic mastoid; enlarged antrum; necrosis of tegmen tympani	None	Uneventful	Mucoid discharge still present

3. Fracture of the processus cochleariformis and removal of the tensor tympani muscle from its semicanal.

4. Removal of the peritubal granulations and necrosis.

5. Burring down of the hypotympanic ledge so as to allow for removal of the pathologic tissue in the hypotympanic cells.

6. Shaving of the facial ridge nearly to the level of the nerve, including careful curettage of the granulations about the shell-like pyramidal process and the round window.

The after-care is relatively simple. No packing is used. We have deviated from the formula of maintaining the mastoid cavity, middle ear and external ear as one. The mastoid cavity is allowed to become obliterated with granulation tissue if it will. The final cavity often appears as a somewhat enlarged external canal and tympanum. To date we have encountered no great difficulties; in fact, the healing is often hastened and is better because of the smaller surface to be epithelized. The caliber of the endaural cavity is dependent on the use of the large radical operating speculum through which the dressings are done. In a few cases more tissue accumulated on the mesial wall than we desired, but no complications have resulted from this. A dry ear has been obtained in 89.6 per cent of the cases.

Conclusions.—It is conceded by many that the endaural mastoidotympanectomy offers definite advantages for the surgeon trained in endaural technic, among which are the following:

1. The operation is aimed at a direct exposure of the usual site of the chronic disease.

2. The immediate exposure of the antrum through the posterior bony canal wall in many cases makes it unnecessary to remove large areas of cortex and often avoids the uncovering of sinus and dura, thus offering a minimal sacrifice of normal tissue.

3. The postauricular mastoidotympanectomy requires a postauricular incision for exposing the antrum and for treatment of possible pathologic conditions in the mastoid plus an endaural incision for the performance of plastic repair and the care of lesions in the middle ear and attic. The endaural approach removes this necessity of a double incision.

4. The operation has resulted in a greater percentage of dry ears than we obtained by any other method.

NOTE.—Since the conclusion of this article, we have performed operations in over 50 cases, bringing our total number to over 125. The last cases are too recent to report; however, they include 1 case of pneumococcic meningitis with cure and 10 cases of the fenestration operation for otosclerosis.

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From the service of Dr. George H. Woodruff, Illinois Eye and Ear Infirmary.

SULFATHIAZOLE IN THE TREATMENT OF SINUSITIS

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SAN FRANCISCO

The purpose of this report is to review the literature relating to the treatment of sinusitis with sulfanilamide and its derivatives and to record the results of treatment in 10 cases of chronic maxillary sinusitis.

The first reference to the use of sulfanilamide or its derivatives locally in the sinuses was made by Childrey,[1] in April 1938. He reported a case of maxillary sinusitis in which the infecting organisms were short chain streptococci and gram-positive bacilli and in which the condition resolved following irrigation with a soluble solution of sulfanilamide.

Two years later Levin [2] reviewed the action of sulfanilamide and its derivatives as used in otolaryngologic practice. He stated that sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) is suggested for the treatment of staphylococcic infections, including bacteremia, osteomyelitis, sinusitis, otitis media and mastoiditis. His conclusions were that sulfathiazole is less toxic than sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) and that its oral administration offers a valuable adjunct to other forms of treatment of pneumococcic and staphylococcic infections of the ear, nose and throat.

Bowers [3] stated that chemotherapy had entered the field of nasal therapy, although there was little literature on its use in sinus infections. He used both sulfanilamide and sulfathiazole orally in very severe sinus infections which seemed to be getting out of control—those of one to six weeks' duration. In some cases roentgenograms of the sinuses were obtained before and after treatment. Antral lavage was used in combination with the administration of the drug. He stated that the results were miraculous, since he saw no flare-up in the sinusitis following the discontinuance of the treatment with the drugs, such as frequently occurs in mastoiditis.

Spink and Paine [4] published a preliminary report dealing with the local use of sulfathiazole. In their summary they stated that : (1)

1. Childrey, J. H.: Sulfanilamide in Treatment of Sinusitis, Bull. Pract. Ophth. **8**:33 (April) 1938.

2. Levin, B. G.: Sulfathiazole in Otolaryngology, Minnesota Med. **23**:410 (June) 1940.

3. Bowers, W. C.: The Treatment of Sinusitis, Virginia M. Monthly **67**:660 (July) 1940.

4. Spink, W. W., and Paine, J. R.: The Local Use of Sulfathiazole in the Treatment of Staphylococcus Infections, Minnesota Med. **23**:615 (Sept.) 1940.

localized staphylococcic lesions in 16 patients were treated by applying sulfathiazole directly to the affected areas; (*2*) the sulfathiazole preparations included sulfathiazole crystals alone and an aqueous suspension of sulfathiazole used as an irrigant and for saturating dressings, sulfathiazole suspended in cod liver oil and, for acutely ill patients, sulfathiazole as administered orally; (**3**) the success of the local use of sulfathiazole was dependent in a great measure on maintaining a lesion free of purulent and necrotic material, an effect accomplished by débridement and frequent irrigation.

Herrell and Brown [5] asserted that it is generally agreed the exact mode of action of sulfanilamide and its derivatives when given orally or parenterally has not been established. No conclusive opinions can be obtained as to the mode of action of these compounds when applied locally to wounds, either infected or uninfected. There is no question that the local application of sulfanilamide and its derivatives has been followed by excellent results in a number of cases; on the other hand, results of the local use of these drugs in the treatment of infections have been irregular. The authors stated that the use of thiazole derivatives in the treatment of postoperative sinusitis has not produced encouraging results. No details were given. Twenty-one cases were presented. Regardless of the fact that the results were by no means uniform, it was felt that continued use of the same procedure was justified.

Knoll,[6] in a study of 431 patients, commented on otitis media, mastoiditis and sinusitis. The pathogenic organism in this group usually was the staphylococcus, with or without the hemolytic streptococcus or the pneumococcus. Sulfathiazole therapy gave favorable results in the treatment of 25 patients suffering from these conditions. The methods of treatment (oral or local) were not included; therefore, no evaluation of the results was possible.

In an article published as recently as April 1941 Turnbull [7] gave a method for the use of a solution of sodium sulfathiazole (5 per cent) intranasally as a spray. Of 47 of his patients with chronic sinusitis, all but 7 improved. No definite conclusions regarding improvement were drawn after the instillation of a 5 per cent solution into the maxillary or the sphenoid sinuses. After the spray was used, however, an increase in drainage was noted, which may be accounted for, as a 5 per cent solution of sodium sulfathiazole has a p_H of about 9, while the normal

5. Herrell, W. E., and Brown, A. E.: Local Use of Sulfamido Compounds in Treatment of Infected Wounds, Proc. Staff Meet., Mayo Clin. **15**:611 (Sept. 25) 1940.

6. Knoll, A. F.: Clinical Observations with Sulfathiazole, M. Rec. **152**:364 (Nov. 20) 1940.

7. Turnbull, F. M.: Intranasal Therapy with Sodium Salt of Sulfathiazole in Chronic Sinusitis, J. A. M. A. **116**:1899 (April 26) 1941.

p_H of the nose is about 6.5 (Fabricant [8]). Turnbull stated that sodium sulfathiazole seems a perfectly safe agent to use as a spray in cases of chronic sinusitis.

PROCEDURE

In an effort to determine whether a solution of sulfathiazole or sodium sulfathiazole had any effect on chronic maxillary sinusitis when injected directly into the antrum, a series of 10 cases was studied. The standard method was used, as follows:

1. Intranasal puncture of the antrum with a no. 16 gage antrum needle under the inferior turbinate (usual preliminary anesthesia) and lavage with physiologic solution of sodium chloride, p_H 6.5.

2. Culture (usual laboratory methods).

Experimental studies of various other solutions were made, as follows:

1. Routine antral lavage with physiologic solution of sodium chloride twice weekly, a 1 per cent solution of sodium sulfathiazole in physiologic solution of sodium chloride being left in the antrum.

2. After six weeks of irrigation no change was noted, and the strength of the solution of sodium sulfathiazole was increased to 5 per cent, then several weeks later to 10 per cent. Under the latter treatment in 1 case the condition resolved rapidly. In this instance a moderate amount of hemorrhage had occurred after puncture, partially filling the antrum. Thus, a solution of sulfathiazole and blood was formed (Spink and Paine [9]).

3. Because of these results, 5 cc. of the patient's whole blood was mixed with 5 cc. of a 10 per cent solution of sodium sulfathiazole (making a 5 per cent solution of the salt) and injected into the antrum. The whole blood when mixed with sulfathiazole did not clot. In 2 cases the condition resolved under this treatment.

The procedure described was found successful in some cases but not in others. It had been suggested by Osgood [10] that a suspension of sulfathiazole might be effective when used locally. This treatment was finally adopted as most successful and used as routine. This is the procedure:

After the use of a number of vehicles, the most practical method was found to be that of mixing sulfathiazole with a lubricating jelly (K-Y, or luzo). This base is slowly soluble in water, and traces of the sulfathiazole powder have been recovered from the antrum as long as two weeks after instillation. Jelly suspensions containing sulfathiazole in 20 per cent and 50 per cent concentrations were used. About 4 cc. of these mixtures was injected into each antrum two or three times weekly, according to the severity of the infection.

At the time of writing the method described has been routine for three months. Both sodium sulfathiazole and sulfanilamide were used,

8. Fabricant, N. D.: The Significance of p_H of Nasal Secretions in Situ, Tr. Am. Acad. Ophth. (1940) **45**:197 (May-June) 1941.

9. Spink, W. W., and Paine, T. R.: Bactericidal Power of Blood from Patients and Normal Controls for Staphylococci, J. Immunol. **38**:383 (May) 1940.

10. Osgood, E.: Personal communication to the author.

but the sulfathiazole seemed more effective. Cultures of the antral washings revealed Staphylococcus aureus or Staphylococcus albus in all except 2 cases. In most instances several varieties of organisms were present. It is impossible to say which were responsible for the infection and which were unimportant secondary invaders (Rantz [11]).

The accompanying table shows the bacteriologic reports of antral cultures, the therapeutic agent, the duration of infection before treatment and the result.

Data on Cases of Chronic Maxillary Sinusitis

Case	Organisms Yielded on Culture of Material Obtained by Antral Lavage	Therapeutic Agent	Duration of Infection Before Treatment	Time Required for Resolution
1	Staph. albus; Str. haemolyticus; Str. viridans	Solution of sodium sulfathiazole (1 to 5%); 10% sulfathiazole jelly (suspension 20%)	9 mo.	Not resolved; large amount of granulation tissue in antrum; referred for surgical intervention after 3 mo. treatment; discharge decreased 75%
2	Staph. aureus	Sulfathiazole jelly (20%)	18 mo.	Resolved in 6 weeks; 3 injections weekly
3	Staph. albus	Solution of sodium sulfathiazole (5%) and blood	6 mo.	Resolved in 2 weeks; 2 injections weekly
4	Staph. albus	Sulfathiazole jelly	3 mo.	Resolved in 2 weeks; 3 injections weekly
5	Staph. albus; long and short chain gram-negative and gram-positive bacilli (anaerobic)	Sulfathiazole jelly (50%)	18 mo.	Resolved in 6 weeks; 2 injections weekly
6	Str. haemolyticus (reported as pure culture)	Sulfathiazole jelly (50%) and sulfanilamide jelly (50%)	24 mo.	Not resolved; treatment difficult; only 1 injection into maxillary antrum; later sulfathiazole jelly applied locally to nose
7	Staph. albus	Sulfathiazole jelly (50%)	6 wk.	Resolved in 1 week; 2 injections
8	Staph. aureus	Sulfathiazole jelly (50%)	3 mo.	Resolved in 2 weeks; 4 injections
9	Staph. aureus	Sulfathiazole jelly (50%) and sodium sulfathiazole jelly (50%)	6 yr.	Not resolved; after 3 mo. treatment, 3 injections weekly, surgical treatment recommended
10	Str. haemolyticus, beta type	Sulfathiazole jelly (50%)	6 mo.	Resolved; odor disappeared after first treatment; 5 injections

In view of the satisfactory clinical results it was decided to seek experimental confirmation of the results in vitro. This work was carried on in the department of bacteriology of the University of California Medical School by Mrs. Marian P. Gratiot and Dr. M. S. Marshall. Bacteriologic experiments were made with sulfathiazole or sodium sulfathiazole in various concentrations in each of four bases: saline solution, whole sheep blood, sheep serum and tragacanth gel. A culture of Staph. aureus, irregularly hemolytic, which was secured from sinus washings, was used as the test culture. In repeated tests, inoculums of 6,000 to 1,000,000 organisms were added to the reagents in each of the four bases.

11. Rantz, L. A.: Personal communication to the author.

The survival of the organisms was followed by several procedures, all of which indicated little or no bacteriostatic action. The slight action seen was exerted more strongly by sodium sulfathiazole, of the two compounds, and in saline solution, of the four bases. Additional conditions tried included tests under oil seals and under reduced oxygen tension, tests in the presence of surface tension depressants, tests with different inoculums and tests in which action was presumably neutralized by paraaminobenzoic acid. Under none of the conditions tested was bacteriostatic action marked. In some cases death of the organisms was observed after one or two weeks. There was no consistent influence of factors which might be expected to alter the results.

COMMENT

A sulfathiazole jelly, 20 per cent or 50 per cent, was an effective therapeutic agent when injected into the maxillary sinus in which there was evidence of chronic maxillary sinusitis.

A suspension of sulfathiazole seemed more effective than sodium sulfathiazole when used clinically.

The average duration of symptoms was ten months, and the average time required for resolution was about three or four weeks.

Clinically, sulfathiazole seemed effective locally in approximately two thirds of the cases in which it was used. The failure appeared to be due to the fact that there was abundant chronic inflammatory tissue lining the antrum, which the therapeutic agent apparently did not penetrate.

Sulfathiazole and sodium sulfathiazole did not seem to exert any bacteriostatic effect in vitro.

No undesirable effects were noted either generally or locally.

Other local uses of sulfathiazole in otolaryngologic practice have been suggested—in chronic otitis media, chronic sphenoiditis, chronic pharyngitis, chronic laryngitis and bronchiectasis. It may also be effective in postoperative care of the nose, throat and mouth, such as that required after tonsillectomy, operations on the sinuses and dental extractions.

SUMMARY

Sulfathiazole applied locally was effective in the treatment of chronic maxillary sinusitis in approximately 70 per cent of the cases. It is suggested that a trial with sulfathiazole is indicated before surgical treatment is advised.

ACKNOWLEDGMENTS

The Winthrop Chemical Co., Inc., and Sharpe & Dohme supplied the sodium sulfathiazole used in the experiments. Cultures were furnished by Dr. Jess Carr, Laboratory, 384 Post Street; the Children's Hospital Laboratory of San Francisco; the Stanford University Medical School Bacteriological Laboratory, and the University of California Medical School Department of Bacteriology. Cases were referred to me by the Central Medical Bureau, State Relief Association.

General Hospital 148, A. P. O. 1104, San Francisco.

Case Reports

CARDIOSPASM

An Improved Esophageal Dilator

EDWIN BOROS, M.D., NEW YORK

The problem of cardiospasm (preventriculosis; achalasia) is one not only of etiology but of management. The cause being controversial, prevention has not as yet been possible. There has, however, been more or less widespread agreement in regard to the best method of treatment, which consists of instrumental dilation. The great majority of patients do well and seldom require operation.

Cure of this ailment is usually obtained, and in my experience it is lasting. However, it should not be assumed that restoration to normal function is always easily or promptly effected. The obstruction of the esophagus takes place at the diaphragm rather than at the cardiac end of the stomach, as is generally thought, and is commonly believed to be a consequence of the failure of the "diaphragmatic pinchcock" [1] to open properly.

It is obvious that roentgenologic study and esophagoscopic visualization are required before actual treatment is undertaken. Only in this way can one eliminate the possibility of any contraindication to instrumentation. It is assumed that a varying degree of dilatation of the organ above the hiatus is to be expected. At times this enlargement reaches enormous proportions, producing not only impairment of nutrition, in addition to the attendant dysphagia, but pressure symptoms in the chest, which add to the patient's discomfort.

The case to be described embodies the worst features of this ailment and presents problems quite unlike those of the type ordinarily encountered.

REPORT OF A CASE

D. W., a man aged 44 and married, had complained of difficulty in swallowing liquids and solids for about eight years. This had become progressively worse, so that eventually it became difficult for him to sleep unless he induced vomiting. The pressure in his chest was annoying. Although a short man, of 5 feet (152 cm.), his weight of 108 pounds (49 Kg.) appeared to belie his statement that he had maintained a steady weight at all times. There was neither anemia nor evidence of vitamin deficiency. During these years various attempts at treatment had been resorted to. Lavage and dilation had been tried, with no improvement.

He was first seen by me, in consultation with Dr. M. L. Wager, on Sept. 9, 1940. A roentgenogram at that time showed an enormously dilated esophagus, which was kinked and tortuous and presented a considerable transverse enlargement (fig. 1). It will be noted that there were some displacement of the mediastinum and evidence of compression of the lung fields, especially on the left.

From the Bronx Hospital.

1. Jackson, C., and Jackson, C. L.: Bronchoscopy, Esophagoscopy and Gastroscopy, ed. 3, Philadelphia, W. B. Saunders Company, 1934.

At this time the patient was in much misery. It was with some measure of confidence that promise of relief was offered him, for the average case of cardiospasm justifies it. Accordingly, esophagoscopic examination was made and the diagnosis of cardiospasm was confirmed. In the subsequent care of the patient it was planned to use the customary pneumatic dilators. After thorough preliminary lavage of the esophagus and the hypodermic injection of 1/100 grain (0.6 mg.) of atropine sulfate and 1 grain of codeine (0.065 Gm.), anesthesia of the throat was accomplished with a 2 per cent solution of pontocaine hydrochloride. The Mosher [2]

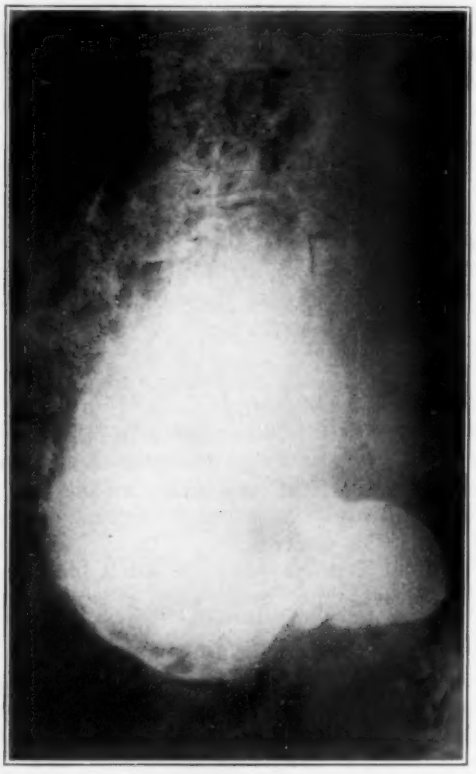

Fig. 1.—Pretreatment roentgenogram of the esophagus.

pneumatic dilator was then introduced under fluoroscopic visualization, but the tip of the instrument could not be made to overcome the sharp transverse bend of the organ, with the result that it remained fixed in the esophageal cavity at about 4 inches (10 cm.) from the hiatus. No better success was experienced with Hurst mercury bougies or with the Tucker [3] pneumatic mercury dilator. Obviously,

2. Mosher, H. P.: Cardiospasm, in Jackson, C., and Coates, G. M.: The Nose, Throat and Ear and Their Diseases, Philadelphia, W. B. Saunders Company, 1929, pp. 1024-1038.

3. Tucker, G.: Cardiospasm: A Pneumatic Dilator, Ann. Otol., Rhin. & Laryng. **48**:808 (Sept.) 1939.

the ordinary means at one's disposal had to be discarded, and if an operation was to be avoided, there was but one recourse—that of dilation of the constriction by means of an endoscopic dilator inserted through an esophagoscope. This would

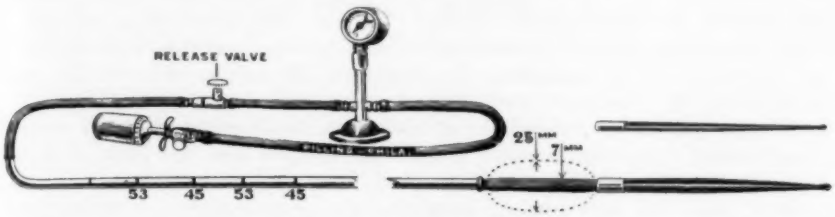

Fig. 2.—Boros-Tucker pneumatic cardiospasm dilator. The flexible tip is detachable and is made in two sizes. Any degree of pressure can be exerted, without fear of perforation.

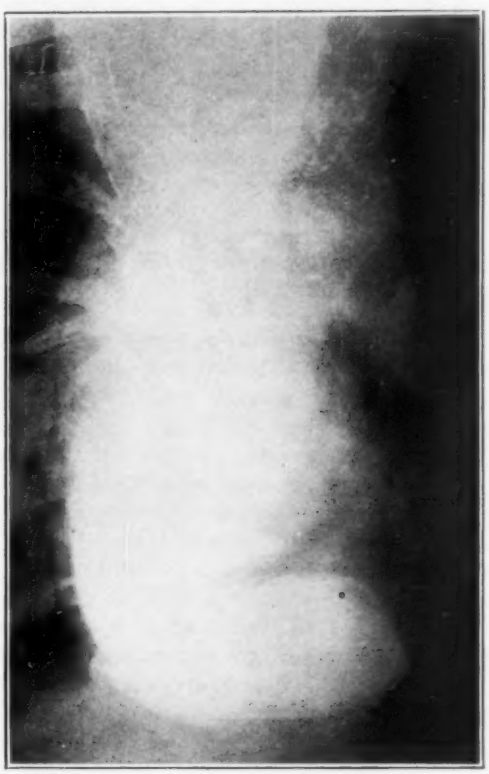

Fig. 3.—Post-treatment roentgenogram of the esophagus.

permit introduction of the dilating instrument into the proper locality under direct vision and with more effectual guidance. In furtherance of this method, a Tucker [3] endoscopic dilator was inserted through a 9 by 53 cm. esophagoscope, but, again,

the sharp turn of the lower end of the esophagus precluded entering the hiatus and attempts at forcing were met with pressure of the opposing wall.

It occurred to me that if the requirements of safety were to be met it would be necessary to resort to a flexible and tapering tip, which might favor finding and entering the diaphragmatic opening. The tip, when attached to any endoscopic dilator, could effect entrance at the desired point and thus make possible the necessary forceful stretching. A detachable silk bougie was used for this purpose (fig. 2). These are made in various sizes to suit one's individual needs.

The new instrument was introduced through an esophagoscope with the patient under careful fluoroscopic control, and it was possible with but little effort to penetrate the diaphragmatic opening when, with further gentle pressure, the required depth had been reached, permitting the much sought-for overdistention of the constriction. This was accomplished by means of the hand bulb, with a pressure registering 5 pounds (2.3 Kg.). The following week the procedure was repeated. Greater improvement in swallowing than had followed any previous attempt at treatment was noted immediately. A roentgenogram taken at this time established definite diminution in the size of the dilated organ (fig. 3), which paralleled the improvement in the patient's subjective symptoms.

COMMENT

Whereas the lesser degrees of cardiospasm are readily amenable to the simpler methods of dilation, exceptions occur in which problems similar to the one in this case may be encountered. Had the instrument described here been unsuccessful in treatment of the patient under consideration, nonsurgical therapy would have been hopeless. Its use may possibly encourage less frequent resort to surgical intervention. Periodic dilations are necessary.

1067 Fifth Avenue.

TYPE III PNEUMOCOCCIC MENINGITIS OF OTITIC ORIGIN

Recovery Following Operation and Treatment with Sulfathiazole and Specific Immune Serum

LEWIS F. MORRISON, M.D., AND EMILE J. GOUGH, M.D., SAN FRANCISCO

The case reported is one of proved severe type III pneumococcic meningitis of otitic origin. The main steps in therapy were surgical drainage, with extensive removal of the original focus of infection and treatment with large doses of sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) and specific type III antipneumococcus rabbit serum.[1]

REPORT OF A CASE

History.—B. S., 36 years of age, a white American, was brought to the University of California Hospital on Aug. 31, 1940 in an irrational and at times semicomatose condition. The history obtained from the referring physician and the patient's relatives was that the patient had been healthy and working at his occupation of hard rock mining until two months prior to the date of entry, when, after swimming in a fresh water pool, a dull pain developed in his right ear. On July 7 he consulted a local physician, who washed out the ear and prescribed the use of ear drops. The drops were used as directed for from three to four weeks. A dull headache and pain in the ear persisted except for short periods of relief. During this time the patient did not feel well but was not bedridden. On August 4 he changed physicians. The second physician incised the right tympanic membrane. The ear drained intermittently until August 12, when it suddenly stopped discharging. The following day the patient noticed a small tender mass below the tip of the right mastoid process. The mass increased in size rapidly and at the end of thirty hours was fluctuant. The mass was incised in the belief that it was an infected lymph node. About 4 cc. of frank pus was obtained. The ear remained dry thereafter, but there was constant drainage of purulent material from the incision below the tip of the right mastoid. The patient's general condition seemed somewhat better in spite of the fact that the dull headaches persisted. On August 29, at noon, he visited the physician to have the dressing over the draining wound changed. He then returned to his home. At 2 p.m. he suddenly became disoriented and vomited. The physician was called. A hypodermic injection was given to quiet the patient. The effect of this administration lasted only a few hours, when the disorientation and vomiting recurred. He was taken to the local hospital, and a spinal puncture was done. Information obtained by telephone was to the effect that the spinal fluid was cloudy and that organisms were found in stained direct smears of the fluid. Other information in regard to pressure and

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From the Department of Surgery, Division of Otorhinolaryngology, University of California Medical School.

1. E. R. Squibb & Sons furnished the sulfathiazole used in this case. The Lederle Laboratories, Inc., furnished the specific antipneumococcus rabbit serum.

laboratory determinations was not obtainable. On August 30 the patient's condition became much worse. Definite stiffness of the neck was evident. He was brought by ambulance some 200 miles (1609 meters) to the University of California Hospital.

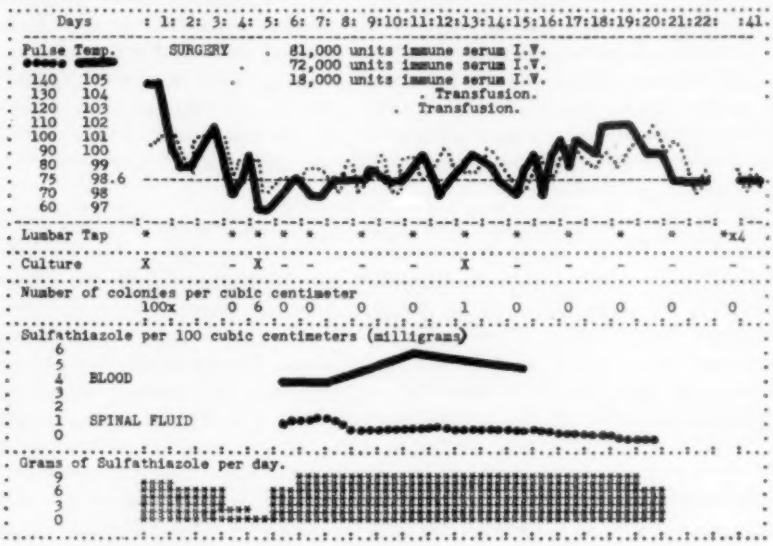
Physical Examination.—The patient was a well developed white man, 36 years of age, who had to be restrained to the sides of the bed by means of cuffs because he was thrashing about, irrational and irritable. He alternated between periods of excitement, during which he muttered unintelligible words, and periods in which he was semicomatose. His temperature was 40.4 C. (104.7 F.) by rectum; the pulse rate was 92, and the respirations were 20 per minute. He was incontinent of urine on entry. The findings on physical examination were marked stiffness of the neck, absence of cremasteric and abdominal reflexes, positive Kernig and Brudzinski tests, negative Babinski and Hoffmann tests and slightly hyperactive tendon reflexes in both the upper and the lower extremities. The pupils were large and fixed to light as a result of the instillation of a solution of an atropine salt by the local physician prior to sending the patient to the hospital. The fundi showed early papilledema of about 1 diopter. There was no evidence of hemorrhage or exudate, and the vessels were within the normal limits. The left auditory canal and tympanic membrane were normal in all respects. There was sagging of the posterior superior wall of the right external auditory canal. The right tympanic membrane appeared to be intact, was slightly bulged and was devoid of the usual landmarks. There was no evidence of discharge in the right external auditory canal. A moderately thick yellow purulent material was escaping from a small sinus tract in the center of a reddened, indurated area approximately 2.5 by 3.5 cm. in size, located just below the tip of the right mastoid process. A diagnosis of mastoiditis on the right side with a draining Bezold abscess and associated meningitis was entered in the record. The duration of the infection, the relatively low grade virulence of the organism up to the last two days, when the acute symptoms appeared, and the stormy course after the acute upset suggested that we might be dealing with a type III pneumococcic infection.

Operative Procedure.—The patient was taken to the surgery and prepared for a simple mastoidectomy on the right side. Ether vapor was used to induce anesthesia. As soon as the patient was relaxed, a spinal puncture was done. The fluid was very cloudy and showed an initial pressure of 560 mm. of water. Approximately 12 cc. was removed. Removal of the mastoid cortex showed a picture typical of long-standing, slowly developing coalescent mastoiditis. There was extensive pneumatization. The cells extended posterior to the lateral sinus, well up into the squamous portion and anteriorly into the root of the zygoma. The tip of the mastoid process was necrotic and was perforated at the lowermost portion. There was an abscess in the soft tissues between the sternocleidomastoid muscle and the posterior belly of the digastric muscle. This abscess was continuous with the draining sinus tract mentioned in the history and in the physical examination. The mastoid cells were exenterated, and the bony walls over the middle and posterior fossae were removed, because we felt that this was a most important step in stopping further feed-in of infective organisms from this area. The dura covering the middle and posterior fossae was tense and moderately inflamed. No visible evidence of a portal of entry in the form of a thrombosed vessel was seen. The wall of the lateral sinus was soft and presented no evidence of infection. This wall was visualized from above the knee to the upper portion of the bulb. The area

was packed wide open with plain tent gauze sponges and the usual dressing applied. At the conclusion of the operation the pulse rate was 115 and the respirations were 24 per minute.

Laboratory Findings,—Examination of the blood on the patient's entry to the hospital showed the hemoglobin content 76 per cent, the red blood cells 3,900,000 and the white blood cells 31,200 per cubic millimeter, with polymorphonuclears 96 per cent.

Cerebrospinal fluid obtained just prior to the operation was under a pressure of 560 mm. of water; it contained 3,883 cells per cubic millimeter, with 76 per cent polymorphonuclears. A stained smear showed numerous gram-positive capsulated diplococci. The Pandy test was markedly positive. Chemical analysis showed protein 41.2 mg. per hundred cubic centimeters of fluid and sugar 0.052 per cent.



Daily treatment and observations on the patient with type III pneumococcic meningitis of otitic origin.

The colloidal gold curve was normal. A culture of the fluid was positive for type III pneumococcus.

A specimen of urine was not obtained on the patient's admission, because of incontinence, but a specimen obtained later was within normal limits.

Postoperative Treatment.—Data on the daily treatment are shown in the chart. A brief summary is presented. The patient was hospitalized for forty days. A lumbar puncture and a simple mastoidectomy on the right, with extensive removal of the bony covering of the middle and posterior fossae, were done within a few hours after entry. Cultures of material from the mastoid area and of the spinal fluid showed a heavy growth of type III pneumococcus. Therapy with sulfathiazole was started on the first postoperative day and continued until the nineteenth postoperative day, when the administration of the drug was stopped because of fever, pain in both ankles, knees and wrist joints and a macular rash on all four extremities. These manifestations, characteristic of drug reaction, started on the seventeenth

postoperative day. There was one break in the continuous therapy with sulfathiazole. On the fourth postoperative day severe urticaria developed and the administration of the drug was discontinued for twenty-four hours. Because it was thought that sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine) might be tolerated, this drug was administered for twenty-four hours. The patient tolerated this drug less well and vomited four times during the twenty-four hour period. Sulfathiazole therapy was resumed but was maintained at a lower level for a few doses and then raised to 9 Gm. daily, in an effort to establish a higher level in the blood and spinal fluid. The reports on the cultures made from the initial specimen of spinal fluid and from material obtained from the mastoid area were not available until the third day after operation. The organism was then definitely established as type III pneumococcus. A total of 171,000 units of specific immune rabbit serum was given intravenously during the fourth and fifth postoperative days. Two small transfusions of 250 cc. of whole citrated blood were given, one on the tenth and the other on the eleventh postoperative day. Sixteen lumbar punctures and modified complete drainage were performed during the period of hospitalization. The amount of fluid obtained varied from 10 to 40 cc. Cultures and chemical studies were carried out with each sample obtained. On the first, fifth and thirteenth days a pure culture of type III pneumococcus was obtained. The spinal fluid was sterile after the puncture on the thirteenth postoperative day and remained sterile thereafter.

Course.—The patient became rational during the first postoperative day and has remained so. By the fourth postoperative day his temperature had returned to a normal level. On the third postoperative day there appeared on the extensor surfaces of the extremities and on the skin over the anterior thoracic region a generalized urticaria with wheal formation. This was accompanied with gradual development of edema of the dorsum of both hands. The administration of 0.5 cc. of solution of epinephrine hydrochloride by hypodermic injection caused the urticaria to disappear for a few hours. It then reappeared and persisted for another twenty-four hours. The course was uneventful until the fourteenth postoperative day, when the patient began to complain of definite arthralgia. The administration of sulfathiazole was stopped on the nineteenth postoperative day, when a macular rash appeared, with an associated rise in temperature. After the discontinuance of sulfathiazole therapy, all signs of complications disappeared. The mastoid wound filled with healthy granulation tissue, and the skin edges of the wound were allowed to approximate. There were no residual neurologic abnormalities. The patient was discharged on the forty-first postoperative day. He has been seen in the outpatient clinic several times since his discharge from the hospital some nine months ago and has remained free of sequelae.

COMMENT

Sulfathiazole was first presented in August 1939, by Fosbinder and Walter,[2] who reported that the action of this drug against pneumococcic infections in mice was "good" when compared with the action of either sulfanilamide or sulfapyridine. An extensive literature dealing with this relatively new chemotherapeutic agent is now available. The reports on

2. Fosbinder, R. J., and Walter, L. A.: Sulfanilamide Derivatives of Heterocyclic Amines, J. Am. Chem. Soc. **61**:2032 (Aug.) 1939.

the experimental work by Long,[3] van Dyke and his associates [4] and others have shown: what may be expected of this drug in the way of therapeutic results; the various manifestations of toxicity [5]; the pharmacologic aspects of sulfathiazole and the efficiency of the drug as compared with that of sulfanilamide and its other derivatives in regard to action on bacteria both in vitro and in vivo; the properties of sulfathiazole,[6] and the indications and contraindications for administration of the drug. In general, the toxic reactions to sulfathiazole are those common to sulfanilamide and its other derivatives except that cutaneous and ocular manifestations are more frequently encountered after sulfathiazole therapy. Kennedy and Finland [7] reported the first case of fatal agranulocytosis resulting from sulfathiazole therapy.

The fact that it is difficult to obtain in the spinal fluid [8] what can be considered a therapeutically efficient titer of sulfathiazole explains why almost all the literature contains warnings against the use of this drug in treating meningitis due to any type of organism. The difficulty in obtaining an effective titer is due to the rapid absorption and excretion of the drug. Late in 1940 Spink and Hansen [5b] reported the successful use of sulfathiazole in a case of pneumococcic meningitis of otitic origin. Their report is the only one we have found in the literature to date which gives credit for recovery from this type of infection to the administration of sulfathiazole. The literature for the early part of 1941 contains reports of recovery from meningococcic [9] and staphylococcic [10] meningitis treated with this drug.

The fact that the patient whose case we are reporting received both specific immune serum and sulfathiazole therapy may seem an insult to the academic purist. No apology is offered. From a practical point of view the poor condition of the patient, due to an apparently overwhelming infection, warranted the employment of any and all available adjuncts to assist him to combat this infection. We adhered as closely as possible to the routine for the treatment of pneumococcic meningitis outlined by

3. Long, P. H.: Sulfathiazole and Sulfamethylthiazole, J. A. M. A. **114**:870 (March 9) 1940.

4. van Dyke, H. B.; Greep, R. O.; Rake, G., and McKee, C. M.: Observations on the Toxicology of Sulfathiazole and Sulfapyridine, Proc. Soc. Exper. Biol. & Med. **42**:410 (Nov.) 1939.

5. (*a*) Rake, G.; van Dyke, H. B.; Corwin, W. C.; McKee, C. M., and Greep, R. O.: J. Bact. **39**:45 (Jan.) 1940. (*b*) Spink, W. W., and Hansen, A. E.: Sulfathiazole: A Clinical Evaluation, J. A. M. A. **115**:840 (Sept. 7) 1940.

6. Lott, W. A., and Bergeim, F. H.: 2-(Aminobenzene Sulfonamide)-Thiazole: A New Chemotherapeutic Agent, J. Am. Chem. Soc. **61**:3593 (Dec.) 1939.

7. Kennedy, P. C., and Finland, M.: Fatal Agranulocytosis from Sulfathiazole, J. A. M. A. **116**:295 (Jan. 25) 1941.

8. Sadusk, J. F.; Blake, F. G., and Seymour, A.: Observations on the Absorption Excretion, Diffusion, and Acetylation of Sulfathiazole in Man, Yale J. Biol. & Med. **12**:681 (July) 1940.

9. Banks, H. S.: Sulfathiazole in Cerebrospinal Fever, Lancet **1**:104 (Jan. 25) 1941.

10. (*a*) Sadusk, J. F., Jr., and Nielsen, A. E.: Use of Sulfathiazole in Staphylococcic Meningitis with Recovery, J. A. M. A. **116**:299 (Jan. 25) 1941. (*b*) Dietel, F. W., and Kaiser, A. D.: Staphylococcus Aureus Meningitis Successfully Treated with Sulfathiazole, ibid. **115**:601 (Aug. 24) 1940.

Finland, Brown and Raugh.[11] We were fortunate in obtaining at the onset the personal attention and timely helpful advice of one of these co-authors, Dr. J. W. Brown. The various phases of the course of therapy are discussed in semioutline form.

1. Lumbar puncture. Sixteen lumbar punctures were performed in the course of thirty-nine days. At first lumbar puncture was done daily, then every other day, and subsequently longer intervals were permitted between punctures. Drainage was not complete in every instance. The spinal fluid pressure and the report of the culture from the preceding specimen of fluid were used as indicators for the degree of drainage that should be obtained.

2. Removal of the focus of infection. From the otologist's standpoint, this is probably the most important step in therapy. All hope of recovery from meningitis of otitic origin, regardless of the type of organism, rests on the early removal of the focus of infection. The orthodox simple mastoidectomy is not sufficient. It is essential that the bone covering the middle and posterior fossae be removed. When this is done properly, the opportunity for subsequent feed-in of infective organisms is reduced to a minimum.

3. Early and continuous use of the chemotherapeutic agent either by mouth or parenterally. Sulfathiazole therapy was instituted on the first postoperative day, with an initial dose of 2 Gm. This dose was repeated in four hours, and then the amount was reduced to 1 Gm., to be given every four hours thereafter. The appearance of signs of toxic reaction necessitated stopping the drug for a short period on the fourth postoperative day and discontinuing its use entirely on the nineteenth day. A question will arise in the reader's mind as to why chemotherapy was continued for almost seventy-two hours after the appearance of signs of toxication on the seventeenth postoperative day. Antiserum had been given on the third and fourth postoperative days. The appearance of the complication some twelve days later could be explained as well by a delayed serum reaction as by a toxic reaction from the drug. The positive culture obtained from the spinal fluid on the thirteenth postoperative day, even though there was only a single colony per cubic centimeter of fluid, warranted a few more days of chemotherapy. Finally, on the nineteenth postoperative day, when symptoms became so severe, the administration of the drug was stopped. The symptoms promptly subsided.

4. Identification of the infecting organism as soon as possible and intravenous administration of sufficient antiserum to establish a balance of antibody in the circulating blood. In this case the infecting organism was identified as type III pneumococcus. Specific immune serum therapy was instituted shortly after receipt of the report of the culture. A total of 171,000 units of specific type III antipneumococcus rabbit serum was given intravenously in three separate doses within a period of twenty-four hours.

5. Maintenance of a moderate fluid intake to insure adequate amounts of spinal fluid for drainage. The maintenance of an adequate fluid intake

11. Finland, M.; Brown, J. W., and Raugh, A. E.: Treatment of Pneumococcic Meningitis, New England J. Med. **218**:1033 (June 23) 1938.

is essential to diminish the possibility of the formation of crystals in the urinary tract.

6. Transfusions whenever necessary to combat the secondary anemia which is to be expected with prolonged chemotherapy. Two transfusions of 250 cc. of whole citrated blood were given. A complete blood count and urinalysis were carried out every other day during the period of chemotherapy.

The accompanying chart shows that the chemotherapy in this case exceeded in amount the recommended dose as presented in the current literature. The scattered occurrence of positive cultures from the spinal fluid, as on the fifth and thirteenth days, necessitated continuation of the chemotherapy. A total dose of 150 Gm. (2,250 grains) was administered. This is more than six times the recommended dose.

The question of the selection of a sulfanilamide derivative naturally presents itself. Sulfathiazole was selected initially because of its relatively low toxicity and the fact that there was no way of being certain of the identity of the invading organism until the laboratory report was available. When the identity of the invading organism was established, the administration of sulfapyridine by mouth was tried for a short period, but this drug was found to be very toxic. It was not given parenterally because it is still thought that this drug reacts both locally on the gastric mucosa and centrally in the medulla. There was no point in deliberately endangering the patient's chances for recovery by insisting on sulfapyridine therapy. We returned to sulfathiazole therapy. The level of this drug obtained in the cerebrospinal fluid, from 0.5 to 1 mg. per hundred cubic centimeters of fluid, was very low and did not at any time represent more than from 10 to 20 per cent of the blood level. We believe, however, that it played a definite part in the recovery. Sadusk and Nielsen [10a] have suggested the intrathecal administration of a 0.5 per cent solution of the sodium salt of sulfathiazole in physiologic solution of sodium chloride in cases in which the oral administration of sulfathiazole does not obtain and maintain an effective titer. The British literature, however, is filled with admonitions against the intrathecal use of the sodium salt because, regardless of the concentration of the drug, the solutions are very irritating to the tissues.[12]

SUMMARY

The recovery of a patient with a proved type III pneumococcus meningitis of otitic origin is reported.

The therapy consisted of surgical intervention, spinal fluid drainage, chemotherapy employing sulfathiazole and the administration of specific antipneumococcus serum. The importance of adequate removal of the focus of infection prior to the institution of chemotherapy is stressed. One hundred and fifty Gm. (2,250 grains) of sulfathiazole was given during a period of nineteen days. A total of 171,000 units of type III antipneumococcus rabbit serum was given intravenously on the fourth and fifth postoperative days.

It is hoped that this report may stimulate further work on the value of sulfathiazole in the treatment of pneumococcic meningitis of otitic origin.

12. Thrower, W. R.: The Present Position of Sulfonamide Therapy, Lancet **1**:67 (Jan. 18) 1941. Banks.[9]

Clinical Notes; New Instruments and Technics

PAPILLOMA OF THE TONSIL AND UVULA

ROBERT HENNER, M.D., CHICAGO

In the course of examinations of the ears, nose and throat in the Sixth Corps Area Army Induction Board at Chicago, the author was impressed with the frequency of papilloma of the tonsil and uvula. This frequency was in contrast to the comparative rarity with which these growths have been recorded in the literature. For example, in 1927 Vidau [1] reported from a survey of the world's literature 16 cases of papilloma of the tonsil. New and Childrey,[2] in 1931, reported 10 cases of papilloma of the tonsil seen among 357 cases of tumors of the tonsil and pharynx observed in the Mayo Clinic from 1917 to 1930, inclusive. In 1938 Frank [3] reported 3 cases of papilloma of the tonsil seen over a period of six years by the department of pathology of Michael Reese Hospital.

Frank further stated that "true tumors of the tonsils, whether benign or malignant, are rare lesions, those of benign type being much less frequently observed than the malignant." Because of the difference between the impression obtained in the army induction examinations and the opinion expressed by the aforementioned authors, it was decided to make a sampling study. It was felt that a fair estimate of the number of papillomas could be determined by enumerating those observed in 25,000 examinations.

It was, of course, impossible to perform histologic examinations during an examination of this type. However, the following criteria were used: 1. The lesions involved only the tonsil proper, the tonsillar pillars or the uvula. 2. In the cases selected the growths were sessile or pedunculated masses not attached by broad bases to the area from which they arose. The size of these growths varied from 0.3 to 2 cm. in diameter. Those from the uvula were usually pedunculated, and the papillomatous lesion was attached to the tip of the uvula by a threadlike connection as much as 2 cm. in length. 3. The growths were all grayish white and had a wrinkled, mulberry appearance, as distinguished from the normal smooth pink oral mucosa. Comparable lesions were seen and examined histologically in the department of pathology of the Illinois Eye and Ear Infirmary. These lesions all proved to be the so-called hard type of papilloma. They consisted of villous projections to the free surface of hyperplastic squamous epithelium,

1. Vidau, G.: Fibroma emorragico della tonsilla destra, Arch. ital. di otol. **38**:583 (Sept.) 1927.

2. New, G. B., and Childrey, J. H.: Tumors of the Tonsil and Pharynx, Arch. Otolaryng. **14**:596 (Nov.) 1931.

3. Frank, I.: Papilloma of the Tonsil—with Report of Three Cases, Ann. Otol., Rhin. & Laryng. **47**:715 (Sept.) 1938.

nourished by a rete peg of connective tissue rich in capillaries. The superficial epithelium showed hyperkeratosis, and the diagnosis in all these cases was papilloma durum.

In 25,000 inductees examined 94 papillomas were seen. They were more or less evenly distributed among the three areas described. This would mean that 1 in 266 men examined showed evidence of papilloma in the described areas. It was believed safe to assume, because of the representative nature of the group, that 0.37 per cent of the population had papilloma of the tonsils, tonsillar pillars or uvula.

CONCLUSION

Because of the extreme frequency with which papilloma durum of the tonsils, tonsillar pillars and uvula occurred in a series of 25,000 routine examinations of the ears, nose and throat performed by an army induction board, namely, 1 in 266, I feel that benign tumors of these areas, particularly papillomas, should be considered as common lesions.

The widespread occurrence should offer support to the irritant theory of the formation of papilloma. The extensive use of mouth antiseptics and tobacco and the less frequent use of alcoholic beverages probably offer the irritant in the formation of papilloma durum.

310 South Michigan Boulevard.

NASOPHARYNGEAL RADIUM APPLICATOR

J. Brown Farrior, M.D., and George A. Richardson, M.D.,
Ann Arbor, Mich.

The nasopharyngeal radium applicator is used in the treatment of conduction deafness resulting from the overgrowth of lymphoid tissue about the pharyngeal orifices of the eustachian tubes, in a manner similar to that described by Crowe and associates.

The applicator consists of a standard 50 mg. radium capsule attached to a pliable copper wire (fig. 1). The brass capsule contains 5 standard 10 mg. radium needles. Each radium needle has a wall thickness of 0.03 mm. of platinum; the brass capsule has a wall thickness of 0.8 mm., with a platinum equivalent of 0.2 mm.; the combined wall thickness is 0.5 mm. platinum equivalent, adequate filtration to remove the caustic beta rays.

The nasopharyngeal radium applicator is inserted through the middle meatus or along the floor of the nose. The insertion is somewhat comparable to the passing of a eustachian catheter. Local anesthesia is usually used; however, in a

From the Department of Otolaryngology, University Hospital.

few young and uncooperative children induction of anesthesia by avertin with amylene hydrate has been necessary. One capsule is placed in each side of the nasopharynx in the position shown in figure 2.

At the University Hospital we have used the aforedescribed applicators since September 1929. We have varied the dose from 25 to 50 mg. hr. to each side of the nasopharynx. We are now routinely employing one application in

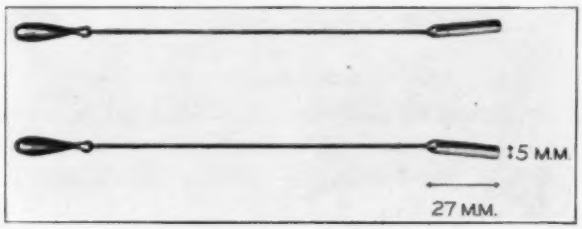


Fig. 1.—Nasopharyngeal radium applicator.

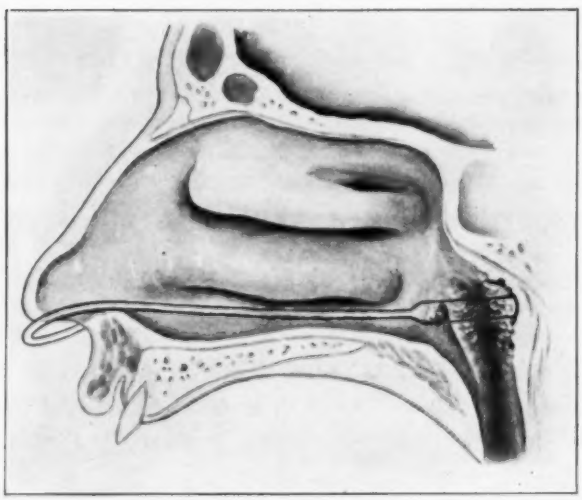


Fig. 2.—Drawing showing the position of the capsule in the nasopharynx, with the base of the capsule over the pharyngeal orifice of the eustachian tube, the center in contact with torus tubarius and the tip resting in the fossa of Rosenmüller.

each side of the nasopharynx for one hour (50 mg. hr.). There have been no undesirable effects. Our results will be reported in a subsequent article.

As compared with the radon applicator, the nasopharyngeal radium type has the disadvantages of being more bulky and requiring a longer period of application. There is one great advantage, however, in that the therapeutic dose of radium is much less expensive, for 50 mg. of radium is available to most otolaryngologists.

Abstracts from Current Literature

Ear

RECOGNITION, TREATMENT AND PREVENTION OF HEARING IMPAIRMENT IN CHILDREN. S. J. CROWE and CURTIS F. BURHAM, Ann. Otol., Rhin. & Laryng. **50**:15 (March) 1941.

The authors are convinced that many of the hearing disorders of adults begin in childhood and that the diagnosis of the condition in early life will do much to prevent subsequent trouble. They feel that 50 per cent of adult deafness can be prevented if school children are examined and treated with this in view. The objective signs and symptoms are: 1. Impaired hearing for high tones (8000; 10000; 13000 and 16000 double vibrations). These values are above the speech range, but as the mucosal changes progress, hearing for tones in this range too becomes involved. 2. Hypertrophic lymphoid tissue in the nasopharynx, involving the eustachian orifice, in the production of mechanical blockage, mucosal congestion in the tube and middle ear, hypersecretion of muscus and, eventually, a myxomatous condition of the mucous membrane or fibrosis, which interferes with ossicular motility. 3. Retraction of Schrapnell's membrane or the entire tympanic membrane. 4. Later, thickening and opacity of the tympanic membrane, so that the mucosa of the middle ear cannot be seen with an otoscope. If these signs are recognized and treatment is instituted before fibrotic changes take place, thus interfering with ossicular movement, much deafness may be prevented.

Adenoids tend to recur in 75 per cent of children if they have been removed before puberty. It is not the size, but the location, of the lymphoid mass which determines whether it is harmful. The two common causes of deafness which can be prevented are suppuration of the middle ear, which is usually secondary to infected lymphoid tissue, and long-continued partial obstruction of the eustachian tubes by hypertrophic lymphoid tissue in the nasopharynx. The studies lead the authors to the conclusion that after the removal of tonsils and adenoids in children latent nodules, which were not evident before, often hypertrophy. They may not obstruct breathing, but because of their location they cause secondary changes in the middle ear. After tonsillectomy and adenoidectomy children should be examined twice a year up to the age of puberty.

Once the situation is recognized as requiring attention, successful treatment is accomplished by a variety of measures: (1) removal of tonsils and adenoids, followed by irradiation to reduce lymphoid tissue which cannot be reached by surgical methods; (2) occasional inflation to remove thick, tenacious mucus in the tube and middle ear and in some cases paracentesis and, with a catheter in the tube, expulsion of the mucus into the external canal, and (3) general hygienic measures, including attention to diet, use of vitamins, change of climate, treatment of allergic tendencies—in short, everything which will improve the health of the child and lessen the frequency of colds. M. V. MILLER, Philadelphia.

AN ANALYSIS OF THE OCULAR FUNDUS CHANGES IN OTITIC SINUS THROMBOSIS. ISADORE FRIESNER, Ann. Otol., Rhin. & Laryng. **50**:32 (March) 1941.

The author reports on the analysis of 25 cases of changes in the fundus varying from blurring of the disk margins to papilledema of as much as 6D. The cases represent about 22 per cent of his cases of sinus thrombosis during the past ten years. His conclusion is that no firm basis for the role of papilledema in sinus thrombosis has been established. He discusses (1) stasis, (2) increased intra-

cranial pressure and (3) toxic states and gives reasons that none of them fulfils etiologic requirements. Inasmuch as the cranial sinuses are part of the dura and the one factor that is common to all these cases is inflammation of the dura, the author feels it is possible that the latter may be an important factor in the development of papilledema in cases of sinus thrombosis.

M. V. MILLER, Philadelphia.

PETROSITIS WITH UNUSUAL COMPLICATION. YOSHIRO YAMADA, Oto-rhino-laryng. **14**:223 (April) 1941.

Yamada reports a case of petrositis in a 27 year old man, a dental student, who presented, in addition to the usual Gradenigo syndrome, a number of unusual symptoms. The patient contracted a light coryza. He complained of headache on the right side, otalgia and tinnitus, pain in the right lower first and second molar teeth and diplopia. Myringotomy relieved for a time his otalgia and headache. The middle ear contained serous exudate. There was impairment of the sensation of taste in the right half of the tongue. Photophobia and lacrimation were present in the right eye. In two weeks this chain of symptoms was followed by suppurative otitis media and acute mastoiditis. The sixth nerve was completely paralyzed, and early signs of meningeal irritation developed. Roentgenograms of the temporal bones failed to disclose involvement of the petrous apexes. On the seventeenth day of illness mastoidectomy was performed—first a simple and then a radical procedure—local anesthesia being used. The operation revealed that the mastoid cortex showed no bony changes. In the mastoid cells there were a small amount of purulent exudate and granulation tissue. Along the superior semicircular canal and deeper in the petrous bone, beneath the dura of the middle fossa, the bone showed an extensive suppurative process. The day following the operation cavernous sinus thrombosis developed, and bilateral exophthalmos, more pronounced on the right. This was followed by death from meningitis. The author deals at length with the pathogenesis and with the symptoms at the various stages of this unusual case. The article is illustrated with two pictures and three diagrams and gives a comprehensive review of the current literature on petrositis.

HARA, Los Angeles.

MAGNESIUM SULFATE SOLUTION FOR TREATMENT OF TINNITUS AURIUM. YOSHIZUMI YAMAMOTO, Oto-rhino-laryng. **14**:232 (April) 1941.

Yamamoto discusses at length the various causes of tinnitus aurium, both the subjective and the objective type. The author treated 14 patients for tinnitus with magnesium sulfate because of the depleting action of this drug on the vascular system. The strength of the solution varied from 5 to 25 per cent. Twenty cubic centimeters was given intravenously every day or every other day, the average number of injections being eleven. In 6 of the 14 patients tinnitus was completely relieved, though 1 of them had recurrence due to mental and physical fatigue. Two patients showed a fair response; four indicated slight improvement, and in 2 there was no change.

HARA, Los Angeles.

Pharynx

APHTHOUS PHARYNGITIS. B. B. BREESE JR., Am. J. Dis. Child. **61**:669 (April) 1941.

Breese, in 1938, observed in a group of children a disease characterized by sudden onset of fever, occasional moderate vomiting and acute inflammation of the throat, without much local soreness. Fever ceased in twenty-four hours, and its disappearance was coincident with the development of a few discrete aphthous

or canker sore lesions in the upper portions of the tonsillar pillars. A similar epidemic was described in May 1939 by Levine, Hoerr and Allanson, and the second minor epidemic was observed in the summer of 1940 by the author. The post-fever lesions (one or more) were yellowish white, slightly raised vesicles 1 or 2 mm. in diameter and usually surrounded by an intense red areola. The lesions were most frequently observed on the faucial pillars, on tonsillar tissue when present and on the soft palate. Sometimes the lesions simulated streptococcic sore throat. The age incidence was from 12 to over 72 years of age.

In some of the patients, petechiae dotted the soft palate and pillars and aphthous lesions appeared later. The absence of pain and discomfort in the throat was in marked contrast to the misery associated with the so-called aphthous, or herpetic, dermatitis in children.

It was decided that the incubation period could not be longer than a week. Results of laboratory studies, including bacteriologic data, were not characteristic.

ROBERT B. LEWY, Chicago.

SECONDARY REPAIR OF CLEFT LIPS AND THEIR NASAL DEFORMITIES. J. B. BROWN and F. MCDOWELL, Ann. Surg. **114**:101 (July) 1941.

Secondary repair of cleft lip and associated nasal deformities may be required though a good primary repair in infancy has been obtained. Prevention of deformity is aimed at in the primary operation by trying to obtain (1) a good alar level and direction, (2) a good nostril floor, (3) a good nostril curve, (4) a straight columella, (5) a full vermilion border without a "whistling deformity," (6) a full lip border in advance of the lower lip and (7) a flexion crease if possible across the lip. It is desirable to have a cupid's bow and a philtrum.

The tendency is to get the lip too long and therefore too narrow, so that the typical retruded lip deformity is produced. The secondary repair usually requires correction of both the lip and the nose. Readjustment of levels, excision of scars and transfer of vermilion-bordered flaps from the lower lip may be necessary. Nasal correction in single clefts may concern reduction of size and reconstruction of the lower lateral cartilages. In cases of double cleft advancement of the columella from the upper lip to give prominence to the nasal tip is often valuable. Orthodontic care of the teeth and the use of a prosthesis to build out the upper lip are occasionally necessary and helpful in restoring good appearance and good function.

HENRY A. MILLER, Philadelphia.

PRIMARY LYMPHOSARCOMA OF THE TONSIL. N. P. COSCO and H. F. POHLMANN, New York State J. Med. **41**:613 (March 15) 1941.

A white man aged 54 complained of "rawness in the throat" of forty-eight hours' duration. There were slight enlargement of the cervical glands, a red throat, and a moderately enlarged right tonsil, from which a biopsy specimen was taken. The Wassermann reaction of the blood was negative. The pathologist's report was "lymphosarcoma of the tonsil." Three radon seeds were implanted in the form of a triangle at the base of the tonsil with use of local anesthesia. Then roentgen therapy was begun. The seeds were removed after nine days. In all, 1,000 millicurie hours of gamma radiation and 3,220 r were distributed over four fields, with one field directed from the opposite side. The patient was last seen about one year later, when examination showed "practical" obliteration of the tonsil and no enlargement of the cervical glands.

The authors stress the point that all this work was done as an office procedure, without the extra expense which hospitalization would have entailed, and the importance of suspecting a malignant condition in similar cases and of making a biopsy.

VOORHEES, New York.

Larynx

SULFANILAMIDE AND SULFAPYRIDINE IN THE TREATMENT OF ACUTE LARYNGO-TRACHEO-BRONCHITIS. R. R. MACGREGOR, Canad. M. A. J. **44**:48 (Jan.) 1941.

In 80 per cent of the author's cases streptococci were cultured from material from the throat. In 1 case of rapidly fatal termination pure cultures of Staphylococcus aureus were grown from material obtained from the larynx and the bronchial tree.

A portion of the summary follows:

"Thirteen cases of acute laryngo-tracheo-bronchitis are reported. Nine cases were treated with sulfanilamide and four with sulfapyridine [2-(paraaminobenzenesulfonamido)-pyridine]. The usual standard dosage was used. As an adjunct blood transfusion was given in all cases on admission to hospital, and the child maintained in an atmosphere of intense humidity. . . . Of the 13 cases 2 died—one in the admitting room of the hospital. The other child, ill for three days, was moribund on admission and died in three hours, the parents refusing tracheotomy. . . ."

Medical treatment to be effective must be given before serious obstruction takes place; so early diagnosis is imperative. MOORE, Omaha [AM. J. DIS. CHILD.].

CARCINOMA OF THE LARYNX: A REVIEW OF ONE HUNDRED AND SEVENTY CONSECUTIVE CASES. W. S. PECK, J. H. MAXWELL and F. LAMBERSON, Laryngoscope **51**:97 (Jan.) 1941.

Peck, Maxwell and Lamberson review 170 consecutive cases of carcinoma of the larynx occurring at the University of Michigan Hospital during the last twelve years. The malignant growth was confined to the vocal cords in 28 per cent of the patients; in 71 per cent it was extrinsic. Practically all of the patients with intrinsic lesions were treated by surgical measures, the small extrinsic lesions by irradiation and the advanced lesions by both methods or not at all. The study led to the conclusion that surgical treatment alone is of value only for intrinsic carcinoma of limited extent, with hemilaryngectomy the operation of choice, and that small extrinsic carcinomas respond comparatively well to irradiation. When doubt exists regarding the purely intrinsic nature of the lesion, irradiation rather than surgical measures should be employed. WOOD, Newark, N. J.

RESULTS OF TREATMENT OF TUBERCULOSIS OF THE TRACHEA AND BRONCHI. JOHN D. KERNAN and A. J. CRACOVANER, New York State J. Med. **41**:482 (March 1) 1941.

The authors have had access to a vast amount of material at the Columbia University Medical Center, at Lenox Hill Hospital and at Seton Hospital. Once a diagnosis of tracheal or bronchial tuberculosis is made, what then? Shall direct examination and treatment be carried out or not? One may have to stretch a scar, remove a small growth or cauterize granulations. Passing a galvanic current through a copper bougie may soften a scar. But some lesions may heal without such local treatment; which? Experience helps somewhat but may be fallacious. The lesions commonly encountered are ulcerations, granulations, fibrous strictures and a swollen mucous membrane. If there is a "wheeze," persistently positive sputum, hemorrhage, severe dyspnea or persistent cavitation, bronchoscopy should be employed unless contraindicated.

As to treatment, diathermy was tried but was given up because of subsequent hemorrhage or excessive scarring. In 21 cases the condition yielded to use of 10 per cent silver nitrate, and in 14, to irradiations with the mercury vapor lamp. The treatment lasted from two weeks to six months. There were 56 cases in all, in 35 of which the patient was clinically cured, in 3 "improved" and in 8 improved but lost sight of; in 1 case the patient was still under treatment, and in 9 the treatment was a complete failure. In 2 cases there seemed to be "spread" of

the infection (?). In 1 of these the lesion eventually healed; in the other the patient died of miliary tuberculosis. One patient died of hemorrhage after cauterization, but autopsy showed that the bronchus was healed. Bronchoscopy is useful, may be urgent and is well worth trying. The mercury vapor lamp may prove to be the most efficient aid in therapy.

VOORHEES, New York.

CARCINOMA OF THE LARYNX IN YOUNG PERSONS: REPORT OF FOUR CASES. E. SAARESTE, Monatschr. f. Ohrenh. **74**:590 (Dec.) 1940.

Of 138 patients with carcinoma of the larynx treated during a period of thirteen years, 13 were under 40 years of age and 4 (2.9 per cent) were under 30. Of these, 3 were women, all with malignant tumor of the vocal cords, while the only male patient in this group had a subglottic carcinoma. The histories and the subsequent treatment of all patients are given in detail, and 9 photomicrographs accompany the article. All 4 patients are regarded as cured, 2 by the local application of radium to the involved cords (with the aid of a window in the thyroid cartilage). One cordectomy (the patient being alive thirteen years after) and one total laryngectomy (on the male, who was living five years after) were successfully performed. The author calls attention to the occurrence of carcinoma of the larynx in the young, particularly in women. Stress is laid on the excellent results of treatment with radium, which gave perfect functional healing; its employment should always be considered in cases of younger persons, in whom preservation of voice is of importance.

LEDERER, Chicago.

Nose

INTRANASAL VACCINE SPRAY: ITS USE IN PROPHYLAXIS AGAINST THE COMMON COLD. THEODORE E. WALSH, Ann. Otol., Rhin. & Laryng. **49**:875 (Dec.) 1940.

The author gives an exhaustive discussion of the etiology of the common cold, the first factor being breaking down of the normal defense mechanism of the nose, which is the mucous covering of the mucosa and the ciliary action, allowing the organisms to enter the subepithelial tissues. Whether or not this develops depends on the presence of pathogenic organisms and the resistance of the organism to them. He discusses the biologic, metabolic and physical factors responsible for the breaking down of the mucous layer and the cessation of ciliary action. He feels that under favorable conditions the nasal defenses are adequate but that conditions may alter the nasal situation, allowing ingress of the infecting agent. Evidence seems to point to a virus as the infecting agent, the secondary invaders being the cause of most of the complications. Factors altering the normal metabolic processes of the body affect the mucosa of the upper respiratory tract by alteration either of ciliary activity and the production of mucus or of the immunologic response of the mucosa. On the physical side he cites the effects of heat on ciliary action and the fact that cilia cease to beat if not kept moist.

The results of subcutaneous and oral administration of cold vaccines having been found unsatisfactory, the intranasal route was investigated. The preparation of the vaccine and the administration by means of an atomizer are described. The results of vaccination by this method of 384 patients over an eight year period are given, and the author expresses his belief that his results warrant the conclusion that intranasal vaccination offers the best prophylaxis.

M. V. MILLER, Philadelphia.

THE IMPORTANCE OF CHRONIC SINUSITIS IN THE TREATMENT OF BRONCHIAL ASTHMA. R. CLARK GROVE, New York State J. Med. **41**:455 (March 1) 1941.

After a review of the literature since Herck, of Freiburg, in 1844, first recognized the importance of nasal pathologic conditions in asthma, Grove analyzes his experiences in 200 cases. Microscopic studies were made of secretions and of

tissues, and in over 85 per cent of cases bacteria were demonstrated. If polyps are present in any of the sinuses, they are best removed by radical operation. Antrotomy (making a window opening) is not satisfactory. Dust and other inhalant allergens must be remembered, both before and after operation. Diet deficiencies, especially an inadequate supply of vitamin A, are to be emphasized. The duration of the postoperative period is important in determining cure; the cases in which improvement occurred represent 69.5 per cent of the 200 cases, on the basis of at least six months' observation. Delay in improvement may be due to foci in the cervical or bronchial lymph glands or in the bronchial mucosa. When treatment, of whatever kind, was "complete" the improvement was 85 per cent, when incomplete, only 54 per cent. There are a "purely infective" and a "combined" type. Allergens and vaccines (autogenous) were used as indicated in the treatment of each type. In management of the strictly antrum types, radical operation (Caldwell-Luc) was about twice as successful as antrotomy.

Grove states that "infective asthma is the result of a sensitization to bacteria or to their products in an individual with an allergic constitution." Each case must be analyzed from all possible medical standpoints.

VOORHEES, New York.

TUBERCULOUS OSTEOPERIOSTITIS OF THE NASAL BONE. F. CHAVANNE, Oto-rhino-laryng. internat. **25**:5 (Jan.) 1941.

Two forms of nasal tuberculosis are recognized: One occurs in young persons and evolves rapidly to a fatal termination; the other is met with in older persons, is more localized and has a favorable prognosis. The disease is rare.

Chavanne encountered the condition in a woman aged 37 years. She had a small fluctuating tumor at the junction of the nasal bone and the superior maxilla on the left side. The front end of the left middle turbinate showed nodules of lupus, and there was a small ulcer on the anterior part of the septum. The cervical glands were enlarged. The Wassermann reaction was negative. Some years previously the patient had had osteitis of the foot. Radiotherapy produced temporary improvement of the septal ulceration. The patient was then given 10 tablets daily of rubrophen (trimethyldioxyoxotritane) of 0.5 Gm. each. This dosage was later reduced to 6 tablets daily. After she took 200 tablets in the course of six weeks, no more of the drug was obtainable. Fistulas had developed at the site of the nasal tumor and in the neck before treatment was begun. The patient improved and gained weight. At the end of six months the adenopathy had diminished and the fistulas had closed.

DENNIS, San Diego.

DISTOMA HEPATICUM IN THE NASAL CAVITY. T. POLYANSKY. Monatschr. f. Orenh. **75**:105 (Feb.) 1941.

A foreign body in the nose of a veterinarian which had been causing "flulike" symptoms and later nasal discharge was removed and found to be Distoma hepaticum.

The infection can be acquired by gnawing grasses, and the lesion is usually observed in the liver. In this case the patient could be regarded as cured with the removal of the foreign body.

LEDERER, Chicago.

Miscellaneous

THE UTILIZATION OF VITAMIN A ADDED TO MINERAL OIL. A. CURTIS and P. HORTON, Am. J. M. Sc. **200**:102 (July) 1940.

The authors seek a method of overcoming the disadvantages attendant on the preferential solubility of carotene in liquid petrolatum, with the subsequent loss of the carotene with the excretion of the oil in the stool. At body temperature 100 cc. of plain liquid petrolatum could hold as many as 466,666 U. S. P. units of

vitamin A in the form of carotene. Amounts of liquid petrolatum as small as 15 or 30 cc., the customary dose of the oil, would still be able to remove all the carotene from a normal diet if the oil came in intimate contact with the food substances containing the carotene. Presaturation of the oil with carotene protected the ingested carotene, but the compound was grossly changed in color and taste and the cost was prohibitive.

Vitamin A concentrate differs from carotene in that it may be utilized readily when fed with liquid petrolatum. The authors fed a mixture of liquid petrolatum and vitamin A to rats showing signs of vitamin A deficiency and found that these rats resumed as good growth as a similar group receiving the same amount of vitamin A in cottonseed oil.

Therefore, one of the major objections to the use of liquid petrolatum as a cathartic can be controlled by making a mixture of vitamin A and liquid petrolatum or by giving vitamin A with the oil. Although carotene would still be lost by its preferential solubility in liquid petrolatum, enough vitamin A in the oil would compensate for this. The authors suggest either taking enough fish liver oil with each dose of liquid petrolatum to supply the daily requirement of vitamin A or making a mixture of liquid petrolatum that would have from 3,000 to 6,000 U. S. P. units of vitamin A in each average dose. This mixture would be colorless and practically tasteless and odorless.

SHAMBAUGH JR. and JENNES, Chicago.

DYSPHAGIA WITH DISORDERS OF THE HEART AND GREAT VESSELS. A. L. BLOOMFIELD, Am. J. M. Sc. **200**:289 (Sept.) 1940.

Dysphagia may occur in the following cardiovascular conditions: dilated left auricle, pericarditis, saccular aneurysm, dissecting aneurysm and anomalies of the aortic arch or of the great vessels springing from it. The difficulty in swallowing is usually related to direct pressure on the esophagus but may be due to spasm via the vagus nerve. Marked compression frequently exists without any interference with deglutition.

While compression of the esophagus by the auricle often occurs, clinical dysphagia from this cause is rare. Besides the case described by the author only 2 instances are to be found in the literature. Dysphagia with pericarditis is a rare event and when present suggests a large effusion. With an aneurysm mild dysphagia, usually due to reflex esophageal spasm, is common and of no serious import; severe, persistent dysphagia suggests a false aneurysm or a huge lesion with threatened rupture into the esophagus and therefore is of considerable diagnostic and prognostic importance. Dysphagia occurs with right-sided aortic arch, double aortic arch and aberrant right subclavian artery. Pressure on the esophagus by the false sac must be a common event in cases of dissecting aneurysm, but clinical dysphagia rarely occurs; dysphagia occurring in a supposed case of occlusion of the coronary vessels should arouse suspicion of a dissecting aneurysm.

SHAMBAUGH JR. and JENNES, Chicago.

THROMBOCYTOPENIC PURPURA DUE TO SULFAPYRIDINE. H. RUSSELL and R. PAGE, Am. J. M. Sc. **200**:495 (Oct.) 1940.

The authors report 2 cases of thrombopenic purpura which developed during the course of treatment with sulfapyridine (2-[paraaminobenzenesulfonamido]-pyridine). The disease was fatal in 1 case and in the other there was also evidence of acute toxic necrosis of the liver, with the appearance of progressive jaundice, without fever or pain, ten weeks after receiving sulfapyridine. The authors suggest that patients receiving sulfapyridine be kept under close observation, with frequent examinations of the blood and urine.

SHAMBAUGH JR. and JENNES, Chicago.

STUDY OF CHANGES IN THE TEMPERATURE AND WATER VAPOR CONTENT OF RESPIRED AIR IN THE NASAL CAVITY. LAUREN E. SEELEY, Heating, Piping & Air Conditioning, June 1940.

Seeley has constructed a rather ingenious apparatus, consisting essentially of a small, thin tube, at the end of which a thermocouple was inserted. The opposite end was connected by a series of tubes and potentiometers, so that it was possible to gage both the temperature and the moisture of air passing through the system. This sampling tube was inserted into the nose at three different positions underneath the inferior turbinate. The tests, on a patient who had an apparently normal nose, were made in a specially controlled, air-conditioned room. Both the inspired and the expired air were sampled at the anterior end, the middle and the posterior end of the inferior turbinate, respectively. From these samples, the author has been able to construct a series of graphs which brought out some rather interesting physiologic relationships. He showed that the inspired air is either cooled or heated at each test position so as to equalize that of the expired air. An increase in temperature may be produced from the air of the lungs and throat. Lastly, partial cooling or reheating of air may occur at the portal of the nose. He found that the loss of moisture from the mucous membrane was influenced by the relative humidity of the inspired air. Tidal air never reaches or leaves the lungs in a saturated condition, irrespective of the temperature and relative humidity of the alveolar air. His tests clearly revealed that the greatest change in temperature and water vapor content take place in the nasal cavity. The nose is the most effective conditioner of respired air. From these studies it is apparent that sudden change of environment may produce an equally sudden change in the rate of loss of nasal moisture. This new rate of loss must ultimately be compensated for by the body if the nasal mucosa is to retain its normal physical characteristics (surface tension, viscosity and osmotic pressure). However, if this adjustment is not made, the nasal mucosa will be at least temporarily altered in its physical characteristics. This, in turn, might permit infection if air-borne infective agencies are present in the new environment.

This study was primarily made from the standpoint of air-conditioning practices and was intended to gage some of the problems that are encountered. Since people pass from one temperature and atmospheric humidity rapidly to another, the condition is equivalent to a climatic change. However, this climatic change is extremely abrupt, and it could not compare with the gradual changes that occur in nature. It has been shown from the tests that the shock of a quick alteration of the environment can be minimized if the indoor relative humidity is controlled in such a way as to prevent any appreciable change in the rate of the loss of moisture from the nasal cavity. The author stresses the fact that air-conditioning engineers must consider the importance of the control of relative humidity, so that the mucous membrane of the entire respiratory tract will not be subjected to any too great alterations and will be permitted to function in a manner as nearly normal as possible.

PERSKY, Philadelphia.

SCLEROMA IN SLOVAKIA. M. SIVAK, Monatschr. f. Ohrenh. **75**:55 (Feb.) 1941.

According to a survey of the literature, the incidence of scleroma seems to be on the increase. A total of 44 cases have been encountered at the Pressburg clinic during a fifteen year period, those of 17 men (36.63 per cent) and 27 women (61.36 per cent). These figures correspond with those given in 2,631 previously reported cases, in which 37 to 41.5 per cent of the patients were males and 54.5 to 58.5 per cent were females. It seems that the incidence is higher in males who are above the age of 35, while women are more prone to be affected when under 30 years of age. In 80 per cent of Sivak's cases hyperplastic changes were found in the larynx—a clinically malignant localization. In 96 per cent of the cases infiltration was observed, and in only 4 per cent, atrophic catarrhal changes. Fifteen of the patients died. Fair success was met in treating the con-

dition with roentgen rays and radium. Less successful was the use of autogenous vaccine and attenuated vaccine. One autopsy is reported in which scleromatous changes were found to involve the entire respiratory tract up to the major bronchi and scleroma bacilli were found in pure culture in the bronchi. The families of 42 patients, in all, 213 persons, were examined. Nearly one third of them showed signs of atrophic rhinitis; the majority of these (2:1) were females. In men the atrophic condition had practically disappeared after the third decade, while in women it persisted. This points to an atrophic susceptibility (*Bereitschaft*) in women. Several curves are shown, to illustrate the relationship of hyperplastic and atrophic changes in scleroma to sex and age. It is suggested that changes in body immunity may explain the appearance of the disease in two antagonistic forms. In Sivak's mind there seems no doubt that a relationship exists between hyperplastic scleroma and atrophic familial catarrh. LEDERER, Chicago.

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

JEROME STRAUSS, M.D., *President*

Regular Meeting, Oct. 6, 1941

Arrested Development of the Larynx Following Irradiation for Recurring Papillomas. DR. SAMUEL SALINGER.

The patient was first seen at the age of 3 years, when he was wearing a tracheostomy tube which had been inserted for relief of dyspnea, supposedly due to diphtheria. Direct laryngoscopy revealed that the larynx was filled with soft papillomas. Before he reached puberty the boy underwent at least forty operations for removal of recurring papillomas, all of which were done under direct laryngoscopic inspection with blunt forceps. At the age of 5 he was subjected to external irradiation with radium, which caused burning of the skin and intralaryngeal swelling, necessitating tracheotomy; a tracheostomy was performed one year later.

When he was seen again after an absence of several years, at the age of 15, the skin over the front of the neck was atrophic and telangiectatic, and the prelaryngeal muscles and the larynx itself had failed to develop. Through frequent use of metal dilators sufficient airway was provided, and it was decided to close the tracheostomy opening; this was done with some difficulty by means of a tubed flap taken from the chest and swung across the neck.

As the boy grew the larynx failed to keep pace, and although a French no. 32 dilator could still be inserted, the airway was inadequate for anything more than simple sedentary pursuits. At present, at the age of 21, a tubed flap is being carried from the back to the neck in order to replace the atrophied skin at the base of the neck, in the hope of releasing the tension on the pretracheal muscles and permitting greater freedom of movement of the larynx. The voice is rather hoarse but is audible in a fairly large room.

DISCUSSION

DR. THOMAS GALLOWAY: I am disappointed that Dr. Salinger did not discuss the mechanism of growth of an organ like the larynx. Cartilage grows interstitially by increase in the number and size of cells and by development of membrane between them, as well as perichondrally. What determines failure to develop after irradiation or injury? Is it injury to chondroblasts or to supporting structures or to both?

Previously, when radium and roentgen rays were used in too great doses or without sufficient screening the radiation did much damage. But Foster has shown that when the rays are used in sufficiently small doses, repeated if necessary, they can destroy the papillomas and not injure the cartilage.

In a case of mine, similar to that of Dr. Salinger's, a boy consulted me with a diagnosis of diphtheria. He had had about twelve operations and some applications of radium. An emergency trachectomy was required, and the larynx was found to be completely filled with grayish masses. I operated on him repeatedly with cup forceps and desiccated the base of the papillomas with diathermy, which seemed to prevent recurrence. From 1929 to 1931 I did everything possible to keep open an airway, including core and tube dilatations, but he did not improve much. He did have a partial airway. Then, about the time of his adolescence

I discontinued treatment, and, much to my astonishment, the larynx started to grow and the airway widened until it was adequate and the laryngostomy opening could be closed; the papillomas did not recur.

I think that in most cases one must blame the operative intervention as well as the irradiation. If one destroys the perichondrium the growth of the larynx will be affected. The use of preparations of "sex hormones" in such cases is interesting, and if its promise is borne out, one may be spared such trials as have been experienced in these cases. They are certainly not common.

DR. PAUL HOLINGER: Dr. Salinger has presented an interesting case. Cases in which observation covers such a long period are extremely instructive. I hope Dr. Salinger will report it in full, for radiologists who treat patients with papilloma of the larynx are pleased with the immediate results obtained in the treatment of the growth but do not see the patient years later, when destruction or failure of growth of the larynx manifests itself. The fallacy of an apparent immediate cure is perfectly illustrated in this case.

DR. GLENN J. GREENWOOD: The case I speak of is that of a boy aged 7 years with recurrent papilloma, whose mother is an intimate friend of a physician I know well. Though everything was going well, the physician desired me to use testosterone propionate. I was loath to do so because the boy was progressing satisfactorily and because the substance had been reported to be of little value in treatment of this condition. Holt had cited 2 cases in which its use was unsuccessful. Nevertheless, I was urged to try it. For about twenty-six weeks the boy has had intramuscular injections of this preparation. For the first seven weeks he received 25 mg. twice a week; for the next seven weeks, 25 mg. once a week, and for the last twelve weeks, 25 mg. every ten days. He weighs, I should estimate, about 50 pounds (22.7 Kg.). When I examined him five weeks ago last Friday the larynx, much to my surprise, was redder throughout and the papillomas previously present appeared to be much paler and firmer. I did not remove any at that time. Last Friday I examined him again, and the picture had changed very little. The papillomas were not growing noticeably. I did, at this time, remove a fragment from the subglottic region. In view of the previous unfavorable reports, I was surprised at the result. At one time an amniotin spray was considered, but this has not been employed because of the good result that is apparently being obtained. His voice has appreciably improved. The size of the genitalia has increased considerably. The administration of the drug is under the supervision of one who is experienced in this field.

DR. ALFRED LEWY: I wish to report 2 cases of multiple papillomas, with apparently spontaneous cure. The first was that of a schoolboy in whom, after several postoperative recurrences, the papillomas spontaneously disappeared, at the age of 15.

The other was that of a youth I first saw at the age of 19. The first surgical removal, which was supplemented by immediate electrocoagulation, was promptly followed by recurrence, with implantations in new areas. After the second partial removal of the recurrent growths the young man entered the University of Arizona, at Tucson, Arizona. I next saw him about six or seven months later. The voice was perfect. There was mild suggestion of an irregularity on one of the ventricular bands, which may have been a remnant of the papilloma.

Both these patients had, as local treatment following operation, a spray of thuja, as recommended some years ago by Fletcher Ingals. In both cases the cure was so slow that I am somewhat skeptical about the influence of the remedy.

DR. FRANCIS LEDERER: As the main premise of Dr. Salinger's paper concerns the stenosis of the trachea, it occurs to me that tracheostomy may have some bearing on the condition. I have two reasons for bringing this up: First, is the possibility that in cases in which tracheostomy was performed in infancy, the stenosis, which occurred low down and was apparently subglottic, was the result of making the opening too high, near the cricoid cartilage. If this was not true in Dr. Salinger's case, it is, nevertheless, known to happen. Second, the history of the patient

was indefinite. He was thought to have had diphtheria. Might not one also assume that tracheostomy could cause a foreign body reaction? The tissue in some of these reactions does not greatly differ histologically from true papilloma. The reason for mentioning this point is that I have witnessed cases in which tracheostomy did act as a foreign body and induce such a local reaction as that described by Dr. Salinger.

DR. WALTER H. THEOBALD: During the development of the larynx, which Dr. Salinger spoke of as being arrested, was there any arrest in development of the mandible? Also, has Dr. Salinger investigated the family characteristics to learn whether there was any hereditary change in the mandible?

DR. SAMUEL SALINGER: The larynx is not fully developed until adult life, and any agent that stunts the growth of the young cartilage cells must certainly interfere with the development of the entire structure. As to stenosis, we did have stenosis at the site of the tracheostomy incision, which, as I stated before, was fully eradicated. There is no stenosis now except for the constriction of the entire larynx, which is not due to webbing or to scar formation. The larynx is merely too small for an adult. If it were scar I should proceed differently, but as the thing now stands no amount of grafting inside the larynx will give more airway.

The important question now is how one would proceed in a similar case if the patient were seen as early as the one presented tonight. It is well enough to say that the patient will outgrow his tendency to formation of papillomas when he reaches puberty. But if a tracheostomy has to be done so the child can breathe, what will happen to the larynx that is not functioning over a period of years? Its development will be delayed. On the other hand, repeated removal of the growths carries the risk of trauma to the delicate mucosa, with possible distortion due to scarring or the development of a stricture. I have had no experience with the use of surgical diathermy, as practiced at the Mayo Clinic, or with the insufflation of estrogenic substance, as recently reported by Broyles. Nevertheless, should I again be confronted with such a case, I should undoubtedly give amniotin a trial.

A New Plastic Flap for Use in Endaural Radical Mastoidectomy. DR. GEORGE E. SHAMBAUGH JR.

The endaural approach for radical mastoidectomy, utilizing the extracartilaginous incision of Lempert, is compared with the postaural approach as follows:

1. Since the infected tissue in a chronic suppurating ear requiring radical mastoidectomy lies, in the majority of cases, largely around the middle ear, attic, aditus and antrum, the endaural approach offers as good or better accessibility for the complete and safe removal of this tissue.

2. The endaural approach causes less trauma to soft tissue and diminishes the danger of perichondritis, since the cartilage is not exposed or cut into, as in the postaural operation.

3. The risk of injury to the labyrinth and facial nerve is possibly less with the endaural approach, which is more direct and closer to these structures.

4. The postoperative disability is less with the endaural approach because of lessened trauma to soft tissue. The external deformity is also less.

5. Most important of all, the completed cavity is more accessible after the endaural incision, since the cavity has been created via the same approach through which it must henceforth be viewed and kept clean. The overhanging edges that frequently occur after the postaural operation are eliminated.

The indications for operation are reviewed, and the technic of the endaural radical mastoidectomy and its modifications with the use of the new plastic skin flap is described.

DISCUSSION

DR. ELMER W. HAGENS: About a year and a half ago Dr. Shambaugh demonstrated the endaural radical mastoidectomy on the cadaver, and shortly after

that the operation was performed on the living subject. Since then I have had occasion to do a small number of operations by this method. I think the use of this flap is a good idea. I was never satisfied with the method of making the older flap. It was a bloody procedure and came at the end of a tiresome operation. It is simple to cut this flap with scissors and turn it back. There is no bleeding, and it covers more bone than the older flap. The endaural approach is more direct and simpler once the technic is acquired, and it certainly gives a more direct route to the antrum and the tympanic cavity. There are cases, however, in which the lesion lies so far posterior to the sigmoid sinus that I wonder whether it would be reached as easily by the endaural approach. It would seem more feasible to use the older method in such cases. In postoperative care the endaural approach is easier, and if there is any obstruction one will find it, whereas with the older method there may be an obstruction in the canal which is not found until later.

Some of the cosmetic results in the endaural operation are not good. When the incision is extended to the suprameatal triangle the scar is in a prominent position.

I have been interested in the evolution of the endaural method. The original technic was to make the incision and then use a burr. Later the burr was not used, and still later the gouge was used over the mastoid process, in a manner similar to that employed when the incision was made behind the ear—the technic apparently going back in some respects to the standard postauricular method.

One has to use the technic that seems indicated. It is difficult to say one is going to do a standard radical or a Bondy operation until one has seen the lesion. Roentgenograms are of help, but occasionally they fail to give an accurate idea of the pathologic condition, or even of the anatomic relations.

Dr. George H. Woodruff, Joliet, Ill.: My associates and I have been performing the endaural operation in our service at the Illinois Eye and Ear Infirmary for about three years. I agree that the endaural route affords definite advantages in the performance of tympanomastoidectomy, as well as atticomastoidectomy. I am much interested in the flap Dr. Shambaugh describes. This procedure, which covers the facial ridge with a healthy skin flap, appears to be of definite value in preventing excessive granulations, thus promoting better and faster healing.

In the Bondy operation another procedure has been used which I believe works equally well or better, that is, the flap employed by Lempert in the fenestration operation. If the attic perforation is not too large, the posterosuperior canal wall can be left attached to the drum membrane, and at the conclusion of the operation this flap can be folded back to line the area of the attic and antrum. This promotes rapid healing, just as it does in the fenestration operation.

Dr. Stanton A. Friedberg: I have used the flap described by Dr. Shambaugh in 4 cases—in 3 Bondy operations and in 1 classic radical mastoidectomy, and I can testify to its effectiveness. Dr. Shambaugh indicated that there is necessity of continuing to spread the endaural incision over a period of several weeks in order to insure against stenosis of the external canal. There is a definite tendency toward healing, with a certain amount of stenosis of the canal, unless this is done. It seems too bad to waste the piece of skin which is removed and discarded when the three endaural incisions are connected, as described by Lempert. In the September 1941 issue of the Archives, page 461, a modification is described by Kettel in which he employs the Lempert incision but uses the entire posterior canal wall to lay over the facial ridge. This is an attempt to eliminate a tendency to stenosis in the canal, which is doubtless due to the removal of part of the wall of the posterior canal in the standard endaural approach.

Presence and Action of Lysozyme in the Nasal Mucus. Dr. Carl E. Cahn Bronner (by invitation).

Local resistance of the nasal mucosa to bacteria is suggested by the fact that the upper parts of the nose contain far less bacteria than might be expected, that

artificially introduced bacteria may disappear quickly from the surface of the mucosa and that accidental and surgical wounds rarely become infected. In addition to the mechanical action of the ciliated epithelium and the drainage, lysozyme (Fleming, A.: *Proc. Roy. Soc., London,* s.B **93**:306, 1922) can be considered a factor in the mechanism of local tissue resistance.

On the basis of the extensive literature on lysozyme, which was reviewed by Thompson (Lysozyme and Its Relation to the Antibacterial Properties of Various Tissues and Secretions, *Arch. Path.* **30**:1096 [Nov.] 1940), some facts concerned with conditions in the nose are discussed. The clearing of a suspension of a sarcina, Micrococcus lysodeikticus, by nasal secretion, methods of estimation and quantitative measurement of the lysozyme content of nasal mucus and saliva and the unchanged lytic power of nasal secretion after storage for nine months, even when dried and exposed to light, are demonstrated.

Lysis of the test bacteria by nasal secretion is inhibited in the presence of an acid reaction at p_H values which may occur during the daily and seasonal swings of the p_H on the surface of the nasal mucosa. In the presence of an acid reaction only the first phase of the lysozyme action takes place, that of adsorption to the bacterial surface, without dissolution of the bacterial cell. The second phase, lysis, occurs immediately after neutralization of the nasal secretion, but by adsorption to the surface and by action of the enzyme on the cell membranes the germs may be killed without being dissolved. Thus, nasal secretions can under certain conditions kill soluble bacteria without dissolving them; on the other hand, some pathogenic bacteria which cannot be dissolved by lysozyme may also be killed.

In the dissolution of bacteria by nasal secretion lysozyme is again freed. It is not used up, as are bacteriolytic amboceptors in the serum, and when more bacteria are added it is again effective. In this way 40,000,000 organisms (M. lysodeikticus) were dissolved eight times in succession by nasal secretion in a 1:32 dilution with solution of sodium chloride. Lysozyme action differs from phage action.

Studies were made on the lysozyme content of nasal secretions in healthy persons. The nasal mucosa taken from the septum and the turbinates, as well as the cartilage of the septum, contains much lysozyme. Daily quantitative measurements were made on one person during one month. After irritation of the nasal mucosa by a narrow strip of stiff blotting paper, a clear liquid trickled from the end of the paper into a glass tube. Saline dilutions of this liquid were tested for their power to dissolve a standard suspension of test bacteria (M. lysodeikticus) and for their power to kill these bacteria. The average dilution for complete clearing of the suspension in twenty-four hours was 1 part nasal secretion in 1,000 to 4,000 parts of solution of sodium chloride. This lowest active concentration varied somewhat from day to day; sometimes it dropped to 1 part in 250; for periods of several days it rose to 1 part in 250,000. Killing of 40,000,000 bacteria brought in contact with various dilutions of nasal secretions for twenty-four hours occurred on the average in dilutions between 1:250 and 1:1,000; the highest killing dilution was 1:16,000. The curves indicating the daily values for lysis and for killing were parallel.

Lysozyme could be obtained without irritation by washing the enzyme off the surface of the nasal mucosa. In order to obtain comparable results from different persons on different days, an apparatus was used whereby saline or buffer solutions were blown against the surface of the mucosa through a capillary tube, enclosed in a glass tube of larger size, the opening of which was pressed against the mucosa. When blown through the capillary against the surface of the mucosa the saline solution returned through the wider tube and was collected in a bulb. In this way an equal surface of the mucosa was washed by an equal amount of liquid in day by day tests. The nasal washings contained enough lysozyme to be tested by the cup method in agar pour plates previously inoculated with test bacteria. This gives no quantitative results but permits estimation of the amount of lysozyme washed off the mucous membrane. Of the 4 healthy persons tested over a period of a month, some showed a higher average lysozyme

content in the nasal washings than others. There were marked daily variations of lytic power in all 4 subjects, the trend of the daily fluctuations being remarkably parallel. All showed a low lysozyme content at the same time, and at another time all had a high content, although 2 were confined to bed and 2 were up and about. There was no relation between excretion of lysozyme and temperature, but there may be a relation to atmospheric conditions connected with a sudden fall or rise of barometric pressure.

In accordance with the studies of Fleming and Hilding, lysozyme disappeared or decreased notably during the first two to four days of a common cold. At that time M. lysodeikticus, sarcinas of other types or spore-bearing rods, which were easily dissolved by secretion from healthy noses, were cultivated from the nasal mucosa or from the mucus; these organisms evidently could not be found in the nose if lysozyme was present. This lack of lysozyme in a running nose holds true only for the common cold; the discharge from running noses of hay fever patients shows a high lysozyme content. Reasons that the absence of lysozyme cannot be considered as proof of a causal connection between the common cold and lysozyme are discussed.

DISCUSSION

Dr. Noah D. Fabricant: These are days of national defense; they are also days of nasal defense. Unlike the current interest in national defense, interest in nasal defense began more than a decade ago with the explanation of the importance of the cilia. In time there were studies on the effect of temperature and drugs and, more recently, work on the significance of the p_H of nasal secretion in situ. Now comes an investigation on lysozyme, which, as Dr. Bronner showed, can conceivably be of practical significance in the future. At present, perhaps, the significance is limited, and one cannot buy lysozyme at the drugstore, in the hope of clearing up nasal problems. Nevertheless, the steps taken in this direction may conceivably lead to another great advance in the knowledge of nasal physiology.

I recall the tremendous enthusiasm with which bacteriophage was once regarded. That interest has not completely died out, although it has been put to sleep until some one can revive it by newer methods. The entire subject of lysozyme may be destined for equal neglect, but time alone will tell.

I was particularly interested in Dr. Bronner's comment on the presence of lysozyme in most persons, and the fact that it is absent during the first three or four days of the common cold. This finding is a challenge to further work. Hilding, some years ago, cautiously advanced a similar thesis. Dr. Bronner has been able to confirm his work. The absence of lysozyme during this period explains why it is so difficult to treat a cold during the first two or three days. It is also interesting that persons with definite vitamin A deficiency show little lysozyme in the nasal secretions. One must guard against erroneous interpretation of this phenomenon. Many persons do not have a vitamin A deficiency, and I feel it would be a mistake to give massive doses of vitamin A to increase the lysozyme content.

I was interested also in the fact that in allergic rhinitis the lysozyme content is high. Perhaps this explains why as a general rule one finds so relatively little secondary infection in cases of allergic rhinitis. I should like to ask Dr. Bronner whether the action of lysozyme is increased by immunization.

I think the members of the society are fortunate in hearing this splendid contribution and I hope that Dr. Bronner's interest in this field will continue.

Dr. Carl E. Cahn Bronner: The lysozyme content of tissues or secretions cannot be increased by immunization with material which contains lysozyme or by using lysozyme which has been more or less purified. There is another question: Can bacterial proteins, after destruction of the bacterial cells by lysozyme, be absorbed by the nasal mucosa so that they act as antigens? I do not know.

In immunization the bacteria must be dissolved in some way in the body in order to act as antigens. Allison has shown that bacteria, after being dissolved by lysozyme, are still antigenic. Bacteriolytic, bactericidal and complement-binding antibodies are formed, but the destruction of the bacteria by lysozyme goes so far that no agglutinins or precipitins appear.

JEROME F. STRAUSS, M.D., *President*

Regular Meeting, Nov. 3, 1941

Development of the Olfactory Nerve and the Nervus Terminalis in Man.
DR. ANTHONY A. PEARSON (by invitation).

The anlage of the olfactory nerve is formed by the growth of fibers and the migration of cells toward the brain. These form a bundle which stretches obliquely upward toward the ventral wall of the forebrain. This bundle constitutes the embryonic olfactory nerve. At the time of the initial attachment of the olfactory nerve to the forebrain there is no indication of an olfactory bulb. A slight protrusion soon develops in the region of the attachment of the olfactory nerve to the brain. This protrusion of the brain is the anlage of the olfactory bulb. The bulb develops as an evagination of the wall of the forebrain. The olfactory bulb enlarges, and the embryonic olfactory nerve spreads out into a layer of fibers which partially covers the surface of the bulb. In its early development the olfactory bulb is directed downward, backward and slightly mediad. As development continues it is gradually directed forward.

The ganglion terminale is formed in part by the migration of cells from the medial border of the olfactory placode. These cells form groups along the medial border of the olfactory nerve. The migration of these cells continues until one end of the ganglion terminale lies in contact with the ventromedial wall of the forebrain. There is some evidence that cells migrate from the forebrain into the ganglion terminale. With further development, the ganglion terminale becomes separated from the forebrain, but the connection with the forebrain is retained by fiber bundles. The roots of the nervus terminalis penetrate the ventromedial surface of the forebrain just caudal to the attachment of the olfactory stalk. The larger and more medially placed bundles course in the brain close to the surface for some distance. Some of these fibers reach the septal region of the brain.

The rostral end of the ganglion terminale gives off a branch which is distributed to the anterior region of the nasal septum. Bipolar ganglion cells migrate from this epithelium and become located along the course of the nerve. Some of these nerve cells retain their connection with the epithelium. Several nerve bundles leave the ventral border of the ganglion terminale and follow the branches of the vomeronasal nerve.

DISCUSSION

DR. WILLIAM F. WINDLE: I knew we should hear an excellent dissertation on the nervus terminalis and the olfactory nerve, for I have been close to Dr. Pearson's work and am familiar with what he has been doing. I should be presuming to try to say anything more about the embryology of these interesting structures. The adult anatomy, too, has been well covered, and I am left with little other than functional considerations. I thought the members of this society would be interested in the meaning of these structures in terms of adult physiology, but here one comes to a dilemma, for it must be admitted little is known.

This work is on the frontier. Knowledge of the development of intrinsic structures of the brain of man is practically nil. The work of Wilhelm His in the last century, summarized in 1904, still stands as the basis of much of the present information; there has been little advance beyond his time. Only in the last few years has progress begun again. Much of the knowledge of the structure and

function of the adult human nervous system depends on working out embryologically the gradually expanding structures, as Dr. Pearson has done here. Investigations of this type, which are careful efforts to explore some of this unknown territory, are of foremost importance. Dr. Pearson has done a splendid job in this study.

It would be speculating to try to state the function of the nervus terminalis or the meaning of Jacobson's organ. I cannot help feeling that the nervus terminalis is not just a transient structure in the embryo, which disappears in the adult. The suggestion that it may be both afferent and autonomic is reasonable. One now has a picture of the nasal mucosa innervated not simply by two nerves, the olfactory and the trigeminal, but by several others. Sympathetic fibers reach it, and parasympathetic neurons come to it from the facial and sphenopalatine ganglions. It is now evident that the vomeronasal and terminal nerves may contribute afferent and efferent fibers. The course of the nervus terminalis into the fore part of the brain, the preoptic area and the anterior part of the hypothalamus is extremely interesting. Here are located important autonomic centers. The hypothalamus is closely related to the hypophysis and is known to be concerned with various sympathetic functions. The nervus terminalis may be an important link in this system. One is faced with a challenge to see just what the meaning of this nerve is—what it does and what role, direct or indirect, it plays in the innervation of the nose.

Dr. Samuel J. Pearlman: It is proper for clinicians to listen to dissertations of this character. The physiology of the nose is to a large extent an unexplored field. A few names stand out. Proetz, an otolaryngologist, is foremost in his investigations, and his recent text should be read by all. Elsberg, a neurologist, leads in studies on olfactometry, a field of which, I am afraid, most otolaryngologists are ignorant.

As I understand it, in man there is a true olfactory system, beginning in the schneiderian membrane and ending in the olfactory bulb with its central connections. Another system, the vomeronasal, starts in Jacobson's, or the vomeronasal, organ, and its nerves end in a so-called accessory olfactory bulb, better named the tuberculum vomeronasale. In man this system is generally atrophic, but Jacobson's organ can be seen occasionally in the adult. Von Ruysch showed pictures of it more than one hundred fifty years ago. It is possible at times to insert a probe to a considerable distance.

The relation of the nervus terminalis to Jacobson's organ has been disputed, but it appears to be definitely settled by various investigators, notably Herrick, Brookover and McKibben, that the relation is one of contiguity and not of function. There is, furthermore, ground for speculation as to the function of the nervus terminalis and its central connections because of its close association with olfactory pathways, but no proof exists today that this function is olfactory.

Dr. Robert Dinolt: I wish to say a few words about the anatomy and pathology of the olfactory bulb, which in general do not receive sufficient recognition. Knowledge of the anatomy is highly important for an understanding of the physiology, as Dr. Pearson and Dr. Windle have pointed out. When one inspects sagittal sections of the olfactory bulb, one finds on its tip a cone-shaped body, which Brunner described in 1923 and called the *Bulbuskörperchen*. It is separated from the actual end of the bulb by a shallow, but clearly marked, sulcus. This body is not accidental as I have been able to observe it in 20 of 22 specimens examined. It is a structure in itself. It contains granular cells and myelin fibers, the latter arranged in bundles. These bundles perforate the glomerular and the external granular layers of the olfactory bulb and attach themselves to the inner myelin fibers of the bulb. I have no opinion as to its function and could not trace it to the terminal nerve, a relation which has been suggested.

In view of recent publications, I wish to draw attention to some observations regarding the pathologic conditions of the olfactory bulb. It has been stated that the presence or lack of the glomerular bodies may indicate the atrophy of the olfactory fibers, on the basis of the idea that the mitral cells are never

affected by regressive changes. But Brunner was able to show that the mitral cells may degenerate even in persons with normal noses. In my material of olfactory bulbs from children and adults I have observed two types of degeneration: one with participation of glia cells and one with shrinkage and damage to the mitral cells alone. The type with participation of the glia was more often encountered, and all stages from increase in the number of satellite cells to neuronophagia and, finally, coffin formation were seen.

There is no doubt that the number of mitral cells may vary to a great extent within normal limits, as I have seen a great amount of normal mitral cells even in old persons, in spite of the presence of regressive changes. That these regressive changes were confined to the mitral cells can be proved by the fact that the ganglion cells of the *Bulbuskerne,* an accumulation of large cells within the center of the olfactory bulb, did not show any changes. It seems that the aforementioned degenerative changes of the mitral cells in the olfactory bulb are the result of a lesion of the olfactory mucosa of the nose, although that cannot be said with certainty, as in a number of cases the nose was apparently normal on inspection and still degenerative changes of the mitral cells were later encountered.

Dr. Walter H. Theobald: It is stimulating to listen to such an excellent and technically clear presentation as this. It would be interesting to have a clinician's viewpoint, and there is here a man who has done considerable work on this subject—Dr. Brunner.

Dr. Hans Brunner: I wish to congratulate the officers of the society on placing this interesting subject on the program. Anatomists and pathologists of past decades have been concerned mainly with the olfactory function of the nose. Thus, in the monumental book of Zuckerkandl one finds a long chapter on the olfactory nerve and its central pathways. Later, after the pathology, symptomatology and treatment of the paranasal sinuses had been worked out, the olfactory nerve was almost forgotten—at least by neurologists. Therefore I consider it a credit to Proetz that he devoted a chapter of his excellent book to the olfactory nerve. However, if I remember correctly, he did not mention the "physiologic" degeneration of the olfactory bulb which was demonstrated tonight by Dr. Dinolt. The interest in the olfactory nerve has not only a theoretic but a practical character. I would remind you of the anosmia following head injuries, the anosmia associated with meningiomas of the olfactory rim, the olfactory aura accompanying tumors of the mesial part of the temporal lobe and, also, the recent studies of Elsberg on the pathologic changes in olfactory function in cases of tumors of the brain in general. In view of all these facts, it is obvious that the olfactory nerve deserves greater interest from the standpoint of the clinician.

Dr. Alfred Lewy: I failed to understand whether the nervus terminalis remains throughout life or whether it is a vestigial structure, like Jacobson's organ.

Dr. Anthony A. Pearson: I want to thank the members of this society for their interest, and particularly Dr. Windle and Dr. Pearlman for their discussions. I am familiar with Dr. Pearlman's paper on Jacobson's organ. I had not planned to study Jacobson's (the vomeronasal) nerve, but it was necessary in order to understand the nervus terminalis. The two nerves fit together like hand and glove. The accessory olfactory bulb in man has recently been demonstrated. It is small in man as compared with the structure in the rabbit, in which the vomeronasal nerve can be easily dissected and followed to its attachment to the brain. In man the nerve is small and difficult to follow.

I do not think any one has dissected out the nervus terminalis and demonstrated its course in the nasal septum of adult man. This nerve is prominent in the human fetus and infant. The anterior branches of the nervus terminalis in the nasal septum form a delicate network, which resembles the intricate plexuses in the intestine. In the oldest fetuses studied for this report the glands in the nasal septum had only begun to develop. It may be that some of the fibers of the plexus are related to these glands.

Transeptal Removal of Chondroma Replacing the Sphenoid Bone and Sella. Dr. Gail R. Soper and Dr. Thomas C. Galoway, Evanston, Ill.

This paper will be published in full in a later issue of the Archives.

DISCUSSION

Dr. Walter H. Theobald: I should like to ask Dr. Galloway the final result in this case and the condition of the patient on the most recent examination. I should also like him to comment on the recurrence of tumors of this type.

Dr. Thomas C. Galloway, Evanston, Ill.: Recurrence of enchondroma takes place only when there is incomplete removal. So far as the roentgenogram shows in this case, there has been no change in the main mass of the tumor. There is a slight thickening of the palate, but it does not seem to amount to anything. The headache is gone, and the general condition is fairly good. The ocular symptoms have not improved.

Many tumors of the pituitary gland are treated with irradiation, and the transeptal, or Chiari, method is an ideal approach for insertion of a radon pack. This particular tumor is probably entirely radioresistant.

Gunshot Wound of the Frontal and Temporal Bones. Dr. Robert Henner.

Two cases of gunshot wound are reported which I treated in the course of my duties as otolaryngologist to the Illinois state penitentiaries. They are presented for discussion because of their otolaryngologic interest. The method of removal of the bullet in each case is reported, with roentgenographic and photographic demonstrations.

DISCUSSION

Dr. Samuel Pearlman: There is little I can add to this interesting discussion of bullet wounds of the skull. Rarely seen in private practice, they are occasionally encountered in public hospitals, especially of the prison type, one of which Dr. Henner attends. Aside from the immediate danger of a gunshot wound of the head, the removal of the bullet when the patient survives frequently offers unlooked-for difficulties. The bullet frequently embeds itself deeply and firmly in bone, where it is surprisingly well tolerated as a rule. Once located it resists removal stubbornly, and a forceps must be firmly placed beyond the equator of the missile if it is to be loosened. In spite of the fact that the bullet often produces no symptoms, the patient frequently wishes to have it removed, a psychologic urge the explanation of which is beyond my understanding. A bullet inside the cranial vault is properly in the province of the brain surgeon, although I remember, with admiration, the skill and ingenuity with which the late George Boot, of this society, successfully attacked many of these problems.

Dr. Thomas C. Galloway, Evanston, Ill.: There was a boy at the county hospital who had shot himself in the forehead three days previously; the bullet made a stellate fracture through the frontal bone, and brain substance exploded through the wound, where it became contaminated. The situation looked hopeless. I coagulated the infected brain tissue with surgical diathermy, and it healed without any trouble.

Dr. G. H. Mundt: The most interesting case of gunshot wound I have seen was that of a lad whose father collected old guns and who was shot with a Leuger pistol by another boy. The bullet, of a steel jacket type, entered 2.5 or 3 cm. posterior to the outer canthus of the eye and passed out at the same point on the other side. The only evidence of injury was slight injection of one eye. The boy was totally blind. One globe contained a great deal of blood; the other had apparently not been injured. Eventually, when the blood absorbed, one could see that the optic nerve of the injured eye was evulsed. The boy was perfectly well except for loss of vision. Loss of vision in the second eye was due to optic nerve atrophy, as observed some months later.

DR. JEROME STRAUSS: There was in my office today a patient who was shot accidentally many years ago and has a bullet in the posterior wall of the left antrum. She says she was shot through the right nostril, since there was bleeding from the right nostril at the time. I have had roentgenograms made for a number of years. There is now no sign of where the bullet entered—no hole in the septum, no scar and no deformity of the nose. She is perfectly well, though every once in a while she feels something should be done about the bullet.

JEROME STRAUSS, M.D., *President*

Regular Meeting, Dec. 1, 1941

Osteomyelitis of the Superior Maxilla: Report of a Case with Recovery Following Chemotherapy. DR. ALFRED LEWY.

Osteomyelitis of the superior maxilla carries a high mortality even under the best surgical management. Therefore in the case to be described, in which the patient was seen very early—so early in fact that there could be doubt as to the diagnosis—it was deemed advisable to try chemotherapy without surgical treatment until definite accumulation of pus or necrosis could be determined. There is considerable difference of opinion in surgical circles as to when surgical intervention is indicated in management of osteomyelitis, but practically all the cases reported are those of involvement of the long bones. Smith (*M. Times* **68**:268 [June] 1940), reporting 45 cases in the Brooklyn Medical Hospital, claimed far better results by awaiting localization before surgical treatment. In addition, patients received chemotherapy and supportive treatment. Calonna (*J. Kansas M. Soc.* **11**:493 [Dec.] 1939) advocated supportive treatment and then drilling for drainage. Many surgeons, however, hold that incision and drainage should be instituted as soon as the diagnosis can be established, the earlier the better.

REPORT OF CASE

G. M., a 7 year old girl, entered the Illinois Eye and Ear Infirmary on June 26, 1940 with a diagnosis of orbital cellulitis and right-sided pansinusitis following a cold two weeks before. Swelling of the right eye began two days previously and was increasing. The left nostril permitted breathing, but the right nostril was completely blocked by swelling and no landmarks were discernible. Mucopus oozed between the folds of the swollen mucosa. The right eye was red, tender and chemosed; the lids were edematous, the edema extending to the lids of the left eye, but the right eye was not proptosed. There was hard swelling of the right side of the nose and maxilla, extending to the malar bone; this swelling felt like dense bone covered by edema. There was no nuchal rigidity, and Kernig's sign was doubtful. The right drum membrane was gray and without landmarks, and there was a slight serous discharge therefrom, but no perforation could be seen.

A blood culture was made, but no report was received. The edema overlying the cheek gave the impression of fluctuation, but puncture with a cataract knife down to the periosteum yielded neither pus nor bony dehiscence. The temperature was 104 F. Washing the right antrum through the natural opening brought only dark, fluid blood. The child's blood was typed in preparation for a transfusion, but this was unnecessary because of the rapid improvement with sulfathiazole (2-[paraaminobenzenesulfonamido]-thiazole) therapy. The blood level of the drug was kept at 7.4 mg. per hundred cubic centimeters for four days, and the therapy was then discontinued because of the general improvement. The first blood count showed 77 per cent hemoglobin, 3,350,000 red cells, 12,200 white cells and 62 per cent neutrophils.

In addition to chemotherapy, the antrum was irrigated through the natural opening, in spite of the absence of landmarks; after the first washing purulent material was returned. Hot dressings were applied, and the infra-red ray lamp was used.

The child was discharged in July 1940, apparently fully recovered. When seen six months later she seemed entirely well, and there were no local symptoms in connection with the nose, sinuses or eye.

The diagnosis in this case may be said not to have been fully established, but the palpable bony swelling on the anterior surface of the maxilla, the extension to the orbital contents and the finding of dark, fluid blood alone on the first irrigation of the antrum make this diagnosis probable, even in the absence of a positive blood culture. At any rate, it offers a reasonable suggestion that surgical intervention may be withheld until the diagnosis is fully established. The roentgenogram showed dense cloudiness of the antral, ethmoid and frontal regions, but no bony necrosis could be made out.

DISCUSSION

DR. JOSEPH C. BECK: I saw this patient two days after her admission, when I made a diagnosis of sinal disease with associated orbital cellulitis, so often seen with edema. I thought the condition might be severe osteoperiostitis but did not think of its being true osteomyelitis of the upper jaw. Of course the actual condition will never be known, for, as Dr. Lewy said, the treatment was successful. Another important fact in the successful outcome in the case is that the two men who were residents at the infirmary were the best men in sticking to a case that I have ever seen. They kept a close watch on the saturation point of the blood as well as on the blood count.

One point was not emphasized sufficiently; it was mentioned that the condition of one ear was acute and that the ear was discharging, but the record states there was bilateral otitis media acuta. At one time involvement of the mastoid was threatened. No operation was performed, however, and both ears are now well. When I first saw the patient, I advocated early opening of the fluctuating subperiosteal abscess; I thought drainage would prevent spread of the infection.

The outcome in this case gives one the hope that by means of the treatment which Dr. Lewy so courageously followed, operation can be avoided in many cases. Dr. Lewy deserves much credit for adhering to this treatment as he did. Or shall I say both he and the patient were fortunate, if the case was one of osteomyelitis?

DR. HOWARD C. BALLENGER: I should like to ask Dr. Lewy what organism was recovered. The few cases of osteomyelitis of the superior maxilla that I have seen have been those of infants, in which fistulas developed or which followed injuries with fracture of the maxilla. In a few cases I have suspected a localized osteomyelitic process in adults following an apical root abscess at some distance from the floor of the maxillary sinus, in which erosion finally occurred.

DR. SHERMAN SHAPIRO: I should like to place another case on record. In the summer of 1938 a young man sustained a crushing injury of the left portion of the maxilla through the agency of a large meat hook. When I saw him about four months after the accident, he had a discharging fistula at the orbital margin, with tearing. Irrigation through the natural opening on this side was followed by discharge of a good deal of pus, and a roentgenogram showed severe involvement of the antrum with some necrosis of the orbital rim. I performed the Caldwell-Luc operation in an attempt to get drainage through the nose, enlarged the external fistula and connected it with the antrum. The nasoantral bony wall was found to be gone, and only mucous membrane remained.

Streptococcus haemolyticus beta was obtained in pure culture. In postoperative treatment sulfanilamide was of no value, either administered locally in powder form or internally. Roentgenograms taken some months later showed that the osteomyelitic process was progressing. At the second operation I took about half the orbital floor and left it open externally. It was kept that way for a year;

at the end of that time a roentgenogram showed that much repair had taken place and the condition of the antrum seemed to be clearing up. So I did a Toti-Mosher operation on the tear sac to insure drainage into the nose. I closed the external fistula, and the patient has been well since last April.

This man was under treatment for more than two years. Nothing seemed efficacious until drainage was established. There is still a little tearing in cold weather. The entire bony wall was gone at the time of operation, but at the time of his discharge it had regenerated. I took out large sequestrums at both operations and now show the final result.

No culture of the teeth was obtained during the course of the treatment. All the devitalized teeth were extracted in the attempt to hasten recovery.

DR. THOMAS C. GALLOWAY: In discussing osteomyelitis of the maxilla one should recall that this bone has very little medullary structure; there probably can be what Dr. Beck called osteoperiostitis, but not osteomyelitis, of the maxilla. Various conditions may lead to this. All have seen cases in which, in spite of anything that could be done the condition progressed to involvement of other cranial bones and death.

Dr. Lewy did not report the organism found, which is important. In a man aged 21, the son of one of the best known pathologists in town, after an antrum window operation advancing infection of this type developed, with a fair amount of sepsis. There was no response to chemotherapy. We obtained a culture of anaerobic streptococcus of the type Melanie has shown to be so important. It is difficult to check, but responds to zinc peroxide. I made an incision through the canine fossa and over the inferior turbinate and injected the drug into this area twice a day. The patient made a startling recovery. I shall soon report 6 similar cases. This organism is very small and slow growing, and routine examination does not reveal it. It seems to me the presence of an organism such as this explains the condition in some cases. Zinc peroxide is so specific that it may prove a remedy of considerable value for this infection.

DR. ALFRED LEWY: As a special instructor of residents and interns, Dr. Beck frequently sees cases before I do. He says I should operate, and I think I should not, and vice versa—but we really seldom disagree.

In reply to Dr. Ballenger concerning the origin of this infection, I paid no attention to the ethmoid sinus, which the roengenogram showed to be as cloudy as the antrum. I have seen a number of cases of orbital cellulitis secondary to infection of the antrum. One was safe in this case in assuming that the antrum was the original focus of infection. I first washed out bloody fluid and then pus from the antrum.

Dr. Shapiro's case is, of course, one of well developed osteomyelitis; my case may have been one of osteoperiostitis. I do not believe one could make a distinction. Nevertheless, I believe the course shows that it is possible in some cases to avert surgical osteomyelitis.

About two years ago Dr. Galloway told me of the success of zinc peroxide in treatment of presumably anaerobic infections. I had heard of its use for treatment of spreading cellulitis of the abdominal wall and was about to try it in a case of gangrenous osteomyelitis of the nose and accessory nasal sinuses, which I reported several years ago, but, unfortunately, that day the patient died of a hemorrhage of the internal maxillary artery. However, I did pass Dr. Galloway's suggestion on to a colleague who had a similar case.

Acoustic Trauma in War and in Peace. DR. HENRY B. PERLMAN.

The importance of the audiometer in detecting early degrees of acoustic trauma is pointed out. Examples of the hearing curve characteristic of this early lesion, an abrupt high tone loss with or without a maximum depression at about 4096 cycles, are shown. Experimental data on the relation of the frequency of the traumatizing tone and the resultant threshold depression are presented. This experiment showed that with tests at octave frequencies, the maximum depression

in threshold was an octave above the frequency of the fatiguing tone and that high frequency sounds were more traumatizing than low frequency sounds of equal intensity. No uniform abrupt high tone loss or dip at 4096 decibels was found in subjects traumatized with pure tones.

The military aspects of acoustic trauma are presented, with special consideration of the shock pulse. This is a peculiar pressure phenomenon produced by bombing, a gun blast and the passage of the nose and base of the moving missile. Quantitative data on the pressures developed by these shock pulses and their duration and propagation are presented and integrated with knowledge of the latent period of the protective middle ear muscle reflex and the manner in which the cochlear end organ works. The acoustic sensations produced and the trauma to the perceptive apparatus are thus explained. The discussion is integrated with observations on injuries to the ear due to the shock pulses from bombs made in Spain during the civil war.

DISCUSSION

DR. C. C. BUNCH: This investigation by Dr. Perlman and his study which appeared in the September 1941 issue of the ARCHIVES, page 429, have been of great interest to me because in several respects they tend to verify the observations made in a paper which I read before the American Otological Society in 1937. For that study my collaborators and I searched for material for two years, carefully scrutinizing each record of the type under discussion, before it was decided that we were on the right track. When audiograms of this peculiar type were discovered, we questioned the patients minutely about the possibility of acoustic trauma. Whenever any one about the clinic was known to have been exposed to loud sounds, we tested their hearing to see whether there was any evidence of hearing loss. Later, we made it a practice to inquire of all men over 35 who came to the clinic whether they had served in the army or navy, and in a surprising number of cases in which a positive answer was obtained this type of hearing loss was found.

The study I presented was less than half completed, for we were unable to establish by experiment a definite cause and effect relation. Dr. Perlman has, in a limited way, attempted to do this. Frankly, I was afraid to expose any one to a very loud sound for a long period for fear the exposure might result in permanent loss of hearing. I have had permanent loss of hearing of this type in both ears for more than twenty years. The loss was due, I think, to a single exposure. I did not wish to lay myself liable for damages.

It has been stated frequently that the audiometer gives no information which cannot be secured by the older tuning fork tests. In this case, however, it appears that while tuning forks in the hands of the older teachers did point out the general character of the loss due to acoustic trauma, the audiogram pictures demonstrate it much more definitely than was possible with tuning fork tests. Otherwise, I think Politzer and other earlier investigators would have defined it more precisely. I believe Dr. Perlman was correct when he stated that the tonal dips may appear at frequencies other than 4096 cycles. In one of my ears the deepest part of the dip comes at 6100 cycles and in the other at 6300 cycles, although both ears show considerable loss at 4096 cycles. In my own case the dips were located by means of a pitch range audiometer. For this reason there can be scarcely any doubt that with an audiometer which produces only the octave tones one is almost certain to miss the dips which come at other than the octave frequencies.

The first lantern slide shows a dip in both ears at 1448 cycles, which would have been missed if only the octave frequencies had been tested. In this case the loss was either congenital or acquired before speech habits were formed.

I suspect, also, that high tone losses of this type may in rare cases come from causes other than acoustic trauma. Dr. Perlman, in his first study, mentioned intoxication, syphilis, trauma to the head and retinitis pigmentosa. I have found them only in conditions which I thought were of congenital origin. The second slide shows two records which were almost identical. One was, I think, of trau-

matic origin and the other congenital. The third slide shows the records of a mother and 5 daughters, 3 of whom showed pronounced loss for high tones with normal hearing for low tones. I think the loss in these 3 persons was congenital, or acquired before speech habits were formed, for all 3 had similar speech defects.

DR. SHERMAN SHAPIRO: Two points associated with this paper should be brought out. In some circles abroad differentiation is made between explosion trauma and acoustic trauma. By explosion trauma is meant that which occurs chiefly in wartime, and occasionally in peacetime as a result of extensive detonation. By acoustic trauma is meant the chronic type illustrated by Dr. Perlman. It is known from autopsies that a bomb blast causes multiple hemorrhages in the lungs, liver and other internal organs, as well as the inner ear. This factor, as much as the noise of the blast, can account for war deafness. From a practical standpoint the differentiation should be made, for in the type occurring in peacetime as a result of exposure to noise, such as that in boiler factories, the deafness is different from that caused by explosives in war.

From Dr. Perlman's experiments, is it not correct to say that the upper tones are more vulnerable and that any trauma will affect this part of the scale first?

DR. ALFRED LEWY: In considering acoustic trauma, the factor of vibrations transmitted through the bony skeleton of the body, as occurs in certain industries, should be mentioned. Such a factor would also involve aviators, who are exposed to tremendous vibratory impulses.

DR. HENRY B. PERLMAN: Dr. Shapiro spoke of the sensitivity of the cochlea to high frequencies. The importance of high frequencies in industrial noise is only part of the picture; the rest is concerned with the brief sound pulses about which little is known and which cannot be analyzed as ordinary sounds. These shock pulses are particularly important in acoustic trauma because of their intensity and brief duration (less than the latent period of the muscle contraction of the middle ear). I think the difference between explosion trauma and acoustic trauma is one only of the degree of injury to the perceptive apparatus, and not of its duration or type. Both the shock pulse of an explosion and the sound wave of a noise produce localized irritation of the cochlea.

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION OF OTOLARYNGOLOGY

KARL M. HOUSER, M.D., *Chairman*

WILLIAM HEWSON, M.D., *Clerk*

Regular Meeting, Nov. 19, 1941

GEORGE L. WHELAN, M.D., *Editor of Abstracts*

Leonardo da Vinci's Contribution to Laryngology, Rhinology and Phonetics. DR. CHARLES J. IMPERATORI.

Barred to his contemporaries, Leonardo da Vinci's contributions to anatomy were first published several centuries after his death. Had they appeared during his lifetime they would have revolutionized the study of anatomy and physiology, and the progress of medicine and surgery would have been greatly advanced. His notes and drawings are now available in books based on a collection preserved at Windsor Castle, the "dell'anatomia," published in 1898 and the "Quaderni d'anatomia," published in 1916. I shall limit my discussion to the sections devoted to the study of laryngology, rhinology and phonetics, with a description of nine drawings and excerpts from the accompanying notes. The difficulties of the student of anatomy in the sixteenth century are dramtically

pictured in Leonardo's own words, and brief reference is made to the prolific contributions of this genius, who was artist, sculptor, engineer, anatomist and architect, as well as to some of the more amusing aspects of his conceptions. Leonardo da Vinci was the first to discover and describe the maxillary and the frontal sinus. He also described the facial muscles and their functions. His anatomic drawings of the larynx, trachea and bronchi are of the earliest recorded. His explanation of voice production coincides in part with present day conceptions. A great debt of gratitude is owed to this man, who overcame all the obstacles to scientific work of the age in which he lived and applied to his studies the accuracy and patience of a great scientist and the skill and insight of a great artist and genius.

DISCUSSION

DR. E. B. KRUMBHAAR: As I have nothing to add to Dr. Imperatori's excellent presentation of this phase of Leonardo's work, I hope you will pardon some more general reflections, for Leonardo has interested me ever since, as a medical student, I learned that he had discovered the moderator band of the right ventricle, which some would like to call "the band of Leonardo." When one considers his outstanding achievements in many fields—physics, mathematics, engineering, architecture, music, astronomy and geology, as well as biology and art, it seems no exaggeration to place him at the very front of the giants of the Renaissance—in fact, one of the greatest specimens that the human race has produced.

The "Mona Lisa" and the "Virgin with Saint Anne," at the Louvre, and the "Last Supper," in Milan, have, fortunately, always been with us to gladden the eye of the amateur, but his other productions, especially his anatomic studies, having been so recently rediscovered, are not as thoroughly appreciated. One has only to compare the beauty and accuracy of the anatomic drawings of the "Fogli" and the "Quaderni" with the crude anatomic productions for at least another century to realize their great superiority.

Leonardo's scientific achievements, as you have heard, have great intrinsic merit, but when one contemplates the environment in which he worked, subject to the whims of patron princes, under the damoclean sword of the Inquisition (the reason perhaps that he wrote in his cryptic right to left manuscript), these achievements acquire even greater luster. To what are they due? It is hard to say whether to put first his keen powers of observation, his superlative power of recording graphically better than by words the results of his observation or the scoring power of his inventive imagination. His persistent diligence in the pursuit of Nature was an equally outstanding attribute.

Yet his path was not an easy one. He was, as Victor Robinson puts it, "the illegitimate son, not only of his parents, but of his age." He had the fresh originality of the Greeks but continued many traditional errors of the Arabs. When he made drawings of what he saw, he made anatomic records that are valuable even today, but to conform to tradition, in his writings, he put pores in the ventricular septum, which he could not have seen, as they were not there. His intellectual speculations were often magnificent; yet at times he contentedly offered physiologic explanations that in the light of contemporary knowledge seem flatulent and childish.

His life was in many ways a tragic one, as one appreciates in Vallentin's sympathetic biography. An illegitimate son, not recognized by his father, disliked by his relatives and by many jealous contemporaries, he worked through triumph and misfortune, to be robbed by circumstance of many of the fruits of his scientific labors. As you have heard, his "Treatise on the Voice" has been lost; his projected anatomic work never saw the light, and his voluminous notes lay scattered for centuries. We still are in ignorance of how many are missing today. Leonardo finally died at Amboise, unappreciated and lonely except for one devoted disciple. Even after death he was not left undisturbed. Twice his bones were disturbed in political upheavals and today are lost, or at best buried with many others in an unrecognizable mass. Yet his figure remains as one of the glories of the human race.

Hearing Aid Problems. Dr. Walter Hughson, Abington, Pa.

The most important factor in fitting a hearing aid is the resistance of the patient. In comparing hearing aids and glasses, it should be recalled that ophthalmology is a thousand years old and this particular branch of otology scarcely twenty-five years old.

Multiplicity of Products.—There are 25 or more varieties for sale, 10 of which have been accepted by the Council on Physical Therapy of the American Medical Association. Such acceptance is of no real value to the patient other than assurance that the product is essentially as represented in advertising matter—the parts are of good quality; a reasonable acoustic gain in volume is offered; the device is devoid of extraneous noise and is of pleasing appearance, with established battery drain and a service guarantee.

Again, a hearing aid, if of any importance, costs $150; a pair of fitted eye glasses, $14. Every patient must be warned that noise, as well as speech, will be increased in volume and that even the best of modern aids is subject to distortion, static and feed-back squeal. Hearing aids cannot be fitted in any way comparable to the prescription of eye glasses. The simpler the control mechanism the better for the patient. An aid with a known characteristic peaked at the high or the low frequencies and controlled only in relation to volume is ideal. Tone controls are of little value so far as the patient's convenience and satisfaction are concerned. The patient's first realization of the quality of his own voice is often the most disturbing factor.

How are hearing aids to be fitted, even in the simplest manner? Thresholds of audibility and intelligibility of speech must be determined for the individual deafened patient. Controlled intensity of the spoken voice is preferable to phonograph records. At least 50 sentences must be used and two thirds of them repeated correctly. Any aid giving a minimum gain of 20 decibels in such a test is acceptable provided this gain places the hearing level in the 20 to 30 decibel sector of the audiogram.

Such a test requires a certain knowledge of qualities and characteristics of hearing aids on the part of the examiner and the availability of a sufficient number of better hearing aids for testing purposes. If two or more aids give equivalent gains, the final choice is the patient's in regard to quality of reproduction, size and appearance of the instrument and weight of battery and ease of operation.

At the present stage of development of this restricted specialty, the average otologist is not, and cannot be, equipped to carry out this procedure. If hearing aids are to become of universal aid to the deaf, centers must be sponsored by national societies, the American Otological and the American Laryngological, Rhinological and Otological Societies. Local state, county and city groups must collaborate. Maintenance of reference centers should be purely on a cost basis. In every instance the control of such centers should be the responsibility of a doctor of medicine and a trained technical staff. The responsibility for selection must be in the hands of the otologist, not the hearing aid salesman, and the otologist must be prepared to have many dissatisfied patients.

DISCUSSION

Dr. Norton Canfield, New Haven, Conn.: Are the patients tested with fitted ear pieces?

Dr. Walter Hughson, Abington, Pa.: A universal type of ear piece is used. Any aid performing satisfactorily under such conditions will be greatly increased in efficiency when an individual mold is secured.

Dr. E. H. Campbell: Do hearing aid companies make tests of this kind?

Dr. Walter Hughson, Abington, Pa.: To my knowledge, no agency is equipped to carry out such a test as I have described.

DR. E. B. KRUMBHAAR: What is the relation between tinnitus and deafness, and what pathologic changes are involved?

DR. WALTER HUGHSON, Abington, Pa.: Tinnitus is a masking sound. When cured, hearing improves directly in relation to the intensity of the tinnitus. The actual pathologic picture in tinnitus is for the most part unknown.

DR. C. M. HOUSER: Should a patient be allowed a test period? How much cooperation should one demand from companies? Who should make the ear molds?

DR. WALTER HUGHSON, Abington, Pa.: The rental system of trial is fair. The fee is applied to the purchase price if the instrument is accepted; otherwise it is forfeited. Competition is now so great that one should expect complete cooperation from all companies. A vacuum tube hearing aid comparable in performance to any on the market today can be built for $19, exclusive of everything but cost of material.

The otologist should make the ear mold, but he has neither time nor knowledge of the technic. He has occasionally to handle cases in which damage has occurred accidentally. The cost of procuring an ear piece is at present slightly more than an otologist's fee for fitting the hearing aid.

DR. WARREN B. DAVIS: What should be the cost of testing for a hearing aid?

DR. WALTER HUGHSON, Abington, Pa.: There is no criterion for this additional cost to the patient. After the otologist has made an adequate diagnostic study of the patient's deafness, the cost for the hearing aid examination should be established purely on the prorated maintenance expense of personnel and equipment involved. There should be no factor of profit.

DR. ORAM KLINE, Camden, N. J.: When should bone conduction hearing aids be prescribed?

DR. WALTER HUGHSON, Abington, Pa.: Only when the bone conduction audiogram shows a relatively normal response through the 2048 cycle frequency. Bone conduction is completely ineffective beyond that point. If it requires high amplification to correct a loss, the tactile factor becomes disquieting. For the prescription of a bone conduction hearing aid no single tuning fork can be used as a measure of competence.

Book Reviews

Eagleton's Index and Abstracts of Literature on Progress in Intracranial Lesions Related to Aural and Nasal Conditions. Published in ARCHIVES OF OTOLARYNGOLOGY, 1925, 1926, 1927, 1928, 1929, 1932, 1937, 1940. Published for Gratuitous Distribution to American and British Otologists in the Interest of Anglo-American Unity, and to Commemorate the Enactment of the Lend and Lease Law—"a New Magna Carta" of Democracy's International Cooperation. By Wells P. Eagleton, M.D. Edition limited to 1,000 copies. Paper. Various pagination, with 2 illustrations. Newark, N. J., 1941.

Wells P. Eagleton has had a brilliant career for over a half century. His numerous contributions, his skill in pioneering, his genius for organization and his mighty spirit of enthusiasm and courage, added to his indefatigable energy, have placed him among the most distinguished otolaryngologists in America. Though very active in all national otolaryngologic societies and serving on the high-ranking committees and as an officer of most of them, he has devoted a large portion of his time to private practice and to his hospitals. He is attending or consultant otolaryngologist to many hospitals and cranial surgeon to several, the total number being nineteen. Numerous citations of merit and various awards have been given Dr. Eagleton in the United States, in the Latin-American countries and in Europe.

He has devoted much time to editorial work and from 1925 through 1940 abstracted such of the literature as contained advances in knowledge of intracranial states in relation to normal or diseased conditions of the ear, nose and throat.

He now presents his "Index," a compilation of an enormous amount of work set forth in a practical and serviceable manner, which will be found useful to every otolaryngologist, neurologist and neurosurgeon who has access to it.

Otolaryngology has advanced since Dr. Eagleton enlarged the frontiers of this specialty by demonstrating a relationship between infection in the ear or sinuses and the development of intracranial complications. He has laid especial stress on the importance of recognizing such relations, with particular emphasis on obtaining a good history. Any therapy directed to the intracranial structures was considered incomplete unless the primary focus of infection in the ear or sinus had been thoroughly eradicated. For many years he has taught at meetings and in his writings that the nose protects the throat, ears, lungs, eyes and brain and its coverings.

The reviewer would be failing in his duty were he to omit Dr. Eagleton's prefacing remarks, which follow:

"'Eagleton's Index' is an assembling of all of the articles and of the thoughts or facts emphasized by me in successive abstracts.

"My purpose in its gratuitous publication is to make accessible to any sincere worker a compilation of the recent literature on the borderline subjects pertaining to otology or rhinology and neurology or neurosurgery, a field of surgical activity which has engaged my efforts during the past twenty-five years. Up to the present time, such a compendium of subjects and evaluation of facts has not been presented in its entirety.

"Since sending the 'Index' to press, on the date of the enactment of the Lease and Lend Law, the pronouncement of the 'Atlantic Charter' carried the United States and Great Britain a step further toward that Union of the English-speaking nations in which lies the hope for a continuation of our Christian civilization.

"President Roosevelt's subsequent declaration 'we Americans have taken our battle stations,' presages that out of the united sacrifies and efforts of the

freedom-building nations of the world, there will emerge a more Christ-like culture in the future.

"With thanksgiving for my country's participation in this struggle for freedom, I rededicate this volume."

This rededication is under date of Nov. 17, 1941 (that of the repeal of the Neutrality Law).

Now that the United States has been drawn into World War II since the attack on Pearl Harbor and other territories belonging to this country, there can be little doubt that this volume, created and prepared by Dr. Eagleton, will do much to help the various units and base hospitals, wherever they may be located, in serving with ever increasing efficiency the needs of the Army, Navy, Marines, Air Force and other delegated authority in the successful prosecution of the war and the saving of the peace.

Nasal Sinuses: An Anatomic and Clinical Consideration. By O. E. Van Alyea, M.D., Assistant Professor, Department of Laryngology, Rhinology and Otology, University of Illinois College of Medicine, Chicago. Price $6.50. Pp. 262. Baltimore: Williams & Wilkins Company, 1942.

The author has conducted a series of clinical investigations on the nasal sinuses during the past several years, at the same time correlating his observations with the anatomy and histopathology. He now presents in book form a comprehensive summary of his studies and has compressed into 262 pages an enormous amount of valuable material.

There are in all 12 chapters, dealing with the following subjects: histopathology, the acute nasal infection, the maxillary sinus, the frontal sinus, the frontal cells, the ethmoid sinus, the sphenoid sinus, sinus disease in children, relation of the sinuses to other organs, other complications and benign growths, allergy and, lastly, general surgical and nonsurgical therapeutic measures.

The author is gifted with a fine literary style, which makes for easy reading, at the same time holding the attention of the reader far longer than many another similar work. The descriptions throughout any chapter are clearcut and distinct, informative and interesting. The chapter on histopathology deserves special praise for its lucid presentation. The photomicrographs are delicate, clear and so skilfully labeled that the reader will enjoy his study of the depicted slides. The bibliography at the end of each chapter is valuable, for it enables the reader to follow any particular line of investigation to its ultimate source. The numerous observations and conclusions of otolaryngologists, here and abroad, are coherently reported in the text matter, adding power to the entirely effective message of the author. The author index at the end of the book carries a total of 359 names. Important data are summarized at the end of some chapters. Other chapters conclude with a discussion under one of the following titles: "Review of the Literature," "Concluding Considerations" or "Comment." Controversial subjects are well dealt with; authorities are accurately cited. Case reports are given in moderate detail to bring to light more forcibly important clinical facts. Roentgenograms and other figures throughout the text are clearly explained, a feature which will aid the reader.

The treatment is outlined along broad principles, and all proved measures are utilized. The entire approach is a sound one. The author has always aimed at exactitude and precision.

The binding, printing and paper are first class.

This textbook is recommended to all otolaryngologists, graduate students, residents in otolaryngology and pathologists for frequent review. It probably is a bit too advanced for medical students but would be valuable for consultation on a certain fine point.

Surgical Treatment of Ethmofrontal Sinusitus by Means of an Osteoplastic Method. By RAUL A. BERGARA. Privately Printed. Buenos Aires: Alfredo Frascoli, 1940.

Bergara reviews the various factors favoring chronicity of frontal sinusitis. He cites Lenhart in discussing the factor of size of the frontal sinus in its relation to chronicity of infection and states that in a sinus having a small transverse diameter, i. e., less than 18 mm., there is rarely opportunity for stasis of secretion. When the diameter of the sinus varied from 18 to 30 mm. stasis occurred in 23 per cent of his cases. Finally, when the diameter of the sinus was greater than 30 mm. stasis occurred in 86 per cent.

Bergara states that surgical intervention should be confined to the endonasal route as a first consideration. This is true whether the sinus is large or small. Furthermore, cure is frequently established by this method in a great many cases; a profuse discharge is often diminished to such an extent that it is of no discomfort to the patient; the procedure is an important step in radical treatment anticipating a difficult and hemorrhagic operation, and, finally, it is a means of decreasing the virulence of the disease. The author then states that if, after this preliminary procedure, a radical operation is still indicated, it should have for its purpose, first, extirpation of the entire mucous membrane and visualization of the entire sinus cavity; second, a good esthetic result; third, the least amount of danger to the patient, and, last, ability to meet any exigency due to anatomic variation in the sinus proper.

The technic is as follows: Prior to operation careful roentgenographic studies of the sinuses are made, so that the surgeon may orientate himself to their extent and possible pathologic condition. When time permits, a careful toilet of the nose is established; that is, attention is given to polyps, deviations of the septum, hypertrophy of the turbinates and associated maxillary infections, and attempts at elimination of all possible septic foci are instituted.

The author operates with the region under local anesthesia, using a 2 per cent solution of procaine hydrochloride with epinephrine hydrochloride (1:1,000) to block the nerves. He also anesthetizes the nasal cavity with packs of gauze or tampons saturated in 10 per cent cocaine hydrochloride, to which epinephrine is added.

The cutaneous incision is a circular one, beginning at the eyebrow, at the external limit of the sinus, sweeping upward and around medially and then down practically to the inner canthus of the eye. The skin is separated and then brought downward and forward. The incision is carried through the periosteum, following the curve of the cutaneous incision. However, the author adds a vertical incision along the lateral side of the nasal bone to join the curve of the periosteal incision at about the juncture of the frontal and the nasal bone. The periosteum is retracted over the lateral side of the nose, thus exposing the ascending process of the maxillary bone. The lacrimal sac and the insertion of the tendons of the muscles of the eye are reflected. He joins these perforations through the bone, and with the periosteum attached, he reflects the bone downward, leaving the attachment to the supraorbital ridge, and thus exposes the entire sinus cavity. He exenterates the mucous membrane of the cavity and probes the nasofrontal duct to establish his orientation. After this he resects the highest portion of the ascending process of the maxilla until he reaches the probe in the nasofrontal canal. In this manner the cells of the agger nasi are destroyed, and the middle turbinate appears clearly in the nasal passage. The nasofrontal duct is widened, but care must be employed to avoid the lamina cribrosa of the ethmoid sinus. The lacrimal sac is then replaced, and a drainage tube is inserted into the sinus, extending through the nasofrontal duct into the nasal cavity. It should be of the largest possible caliber and should be fastened more or less firmly in situ, so that drainage may be continuous for twenty-five to thirty days. Should there be a tendency for the secretion to coagulate at the lower end of the drainage tube, it should be cleaned by means of a flexible sound. Then the osteoplastic flap is replaced; the periosteum is carefully approximated and sutured, and the cutaneous incision is closed with

small interrupted sutures. Should a small abscess form at the deeper angles of the suture, it can be opened and a filiform drainage inserted.

In closing, the author reports 18 cases in which excellent results have been obtained both from the esthetic and from the therapeutic standpoint, and he repeats that the sinus should first be treated through the endonasal route and that if a more radical procedure is indicated, the osteoplastic method is the one of choice. The latter procedure is also recommended in cases of chronic fronto-ethmomaxillary sinusitis, but here transmaxillary ethmoidectomy according to Ermiro de Lima's technic precedes the osteoplastic operation on the frontal sinus.

Diseases of the Nose, Throat and Ear: A Handbook for Students and Practitioners. By I. Simson Hall, M.B., Ch.B., F.R.C.P.E., Surgeon to the Royal Infirmary, Edinburgh (Department for Diseases of Nose, Throat and Ear); Lecturer in Diseases of Nose, Throat and Ear. Second Edition. Price $4.50. Pp. 464, with 74 illustrations. University of Edinburgh. Baltimore: Williams & Wilkins Company, 1941.

This textbook, though a "little" book, covers the entire field of otolaryngology, including bronchoscopy and esophagoscopy. It totals in all 446 pages of well written material, 74 illustrations and 4 colored plates. The entire format is first class, and the arrangement of this text is similar to that of the various texts which have been written by eminent British otolaryngologists during the past quarter century.

The illustrations serve emphasis on methods of examination, diagnosis in general and procedures in treatment. There are a number of schemas and diagrams that help no little toward a better understanding of anatomic relations of the structures under consideration.

Near the end of the text is a section consisting of 5 pages devoted to the general subject of formulas, which is most helpful in treating any given condition and which will be found valuable.

This textbook is recommended to all medical students and general practitioners, but because of the wide scope and large amount of material compressed in but 446 pages, it should form good collateral reading for graduate students in otolaryngology, residents and otolaryngologists.

Directory of Otolaryngologic Societies *

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Gordon F. Harkness, 215 Main St., Davenport, Iowa.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia.
Place: Atlantic City. Time: June 8-12, 1942.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Ralph I. Lloyd, 14–8th Ave., Brooklyn.
President-Elect: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Chicago. Time: Oct. 11-15, 1942.

American Broncho-Esophagological Association

President: Dr. W. Likely Simpson, 899 Madison Ave., Memphis, Tenn.
Secretary: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago.
Place: Hotel Dennis, Atlantic City, N. J. Time: June 8-9, 1942.

American Laryngological Association

President: Dr. T. E. Carmody, 277-16th St., Denver, Colo.
Secretary: Dr. Charles J. Imperatori, 108 E. 38th St., New York.
Place: Atlantic City, N. J. Time: May 25-27, 1942.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. James A. Babbit, 1912 Spruce St., Philadelphia.
President-Elect: Dr. James G. Dwyer, 375 Park Ave., New York.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Place: Atlantic City, N. J. Time: June 1-3, 1942.

SECTIONS:

Eastern.—Chairman: Dr. Henry P. Johnson, 32 Deering St., Portland, Maine.

Southern.—Chairman: Dr. William L. McDougall, 915 Doctors' Bldg., Atlanta, Ga.

Middle.—Chairman: Dr. William E. Sauer, 3720 Washington Blvd., St. Louis, Mo.

Western—Chairman: Dr. Emil F. Tholen, 1136 W. 6th St., Los Angeles.

American Otological Society

President: Dr. E. M. Seydell, 107 W. Douglas Ave., Wichita, Kan.
Secretary: Dr. Isidore Friesner, 101 E. 73d St., New York.
Place: Atlantic City, N. J. Time: May 28-29, 1942.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.